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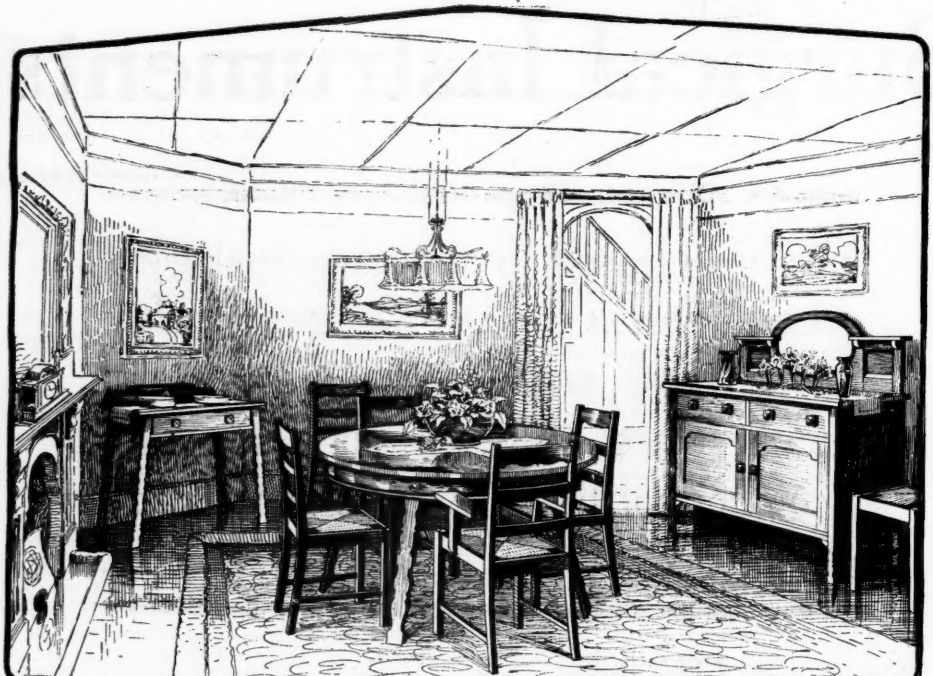

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CLINICAL SYMPTOMS OF ENLARGEMENT OF THE SPLEEN IN CHILDREN.[1]

BY H. DOUGLAS STEPHENS, M.D., M.S. (MELBOURNE),
Honorary Surgeon, Children's Hospital, Melbourne.

IT is my privilege to present for your consideration to-night a short paper on the clinical features of enlargement of the spleen in infancy and childhood.

Anyone who has carefully examined a large number of infants and children, must have been struck by the frequency with which some enlargement of this organ is present. It is, of course, functionally more active in early life and is more often enlarged in the anæmias of early infancy than in later childhood. In many cases the ætiology is obscure or suggestive only, but in others a definite cause is ascertainable or, at any rate, a clinical entity is identifiable. So often is splenic enlargement linked up with disease of the hæmopoietic system that consideration of the one without due reference to the other would render any conclusion nugatory. In fact, one might say definitely that most of the splenomegalies of children do not represent a primary disease of the spleen, but occur as part of other more or less well recognized affections.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 4, 1922.

Clinically the only lesion of the spleen of importance is enlargement and to demonstrate this, palpation is the only method worthy of consideration. Percussion is of little use and often misleading.

Splenic enlargements in children may be conveniently grouped for the purpose of general description under five headings: (*i.*) Those occurring in the course of the acute infectious diseases; (*ii.*) those occurring as the result of so-called constitutional conditions, such as tuberculosis, syphilis, rickets, etc.; (*iii.*) those due to hepatic, cardiac and pulmonary diseases; (*iv.*) those associated with the anæmias, *e.g.*, leuchæmia, splenic anæmia, etc., and (*v.*) such conditions as abscess, cysts, neoplasms, etc., of the spleen.

In order to keep the clinical aspect paramount, it is my intention to attack the subject from a slightly different angle and to discuss it from the point of view of age incidence. I shall consider splenic enlargement, therefore, in three age periods. Firstly, under six months; secondly, between six months and four years; and, finally, from four years of age to puberty.

I have no wish to convey the impression that the diseases listed as occurring in these different groupings are in any way confined definitely to them. Many of the ailments are traceable through all three periods and others again can originate at any age. Only those whose incidence is most pro-

nounced, will be dealt with under the three headings already enumerated.

I. Under six months of age: (1) Syphilis. (2) Tuberculosis. (3) Congenital general œdema. (4) Congenital leuchæmia. (5) Polycythæmia. (6) Anæmia. (7) Acute infections: sepsis. (8) Congenital atresia of bile ducts. (9) Acholuric familial jaundice (familial hæmolytic splenomegaly). (10) Gaucher's disease. (11) Grave familial jaundice of the new-born. (12) Floating spleen.

II. Between six months and four years of age: (1) Deficiency and nutritional diseases. (2) Von Jaksch's anæmia. (3) Leuchæmia.

III. From four years of age to puberty: (1) Acute infective diseases. (2) Chronic heart disease. (3) Hodgkin's disease. (4) Splenic anæmia (Banti). (5) Lardaceous disease. (6) Still's disease. (7) Biliary cirrhosis (Hanot). (8) *Anæmia gravis* (Poynton, Thursfield and Paterson). (9) Sarcoma, cysts, abscess. (10) Tropical diseases: malaria, kala-azar, anchylostomiasis.

The First Age Period.

In an infant under six months in whom an enlarged spleen has been detected, try to ascertain whether the baby was born with it, whether any special features, such as dyspnœa, fever, hæmorrhages, wasting, etc., have been noted and particularly observe and inquire as to the complexion of the infant, whether constantly normal, red, yellow, bluish or pale. It is well to ask if any other member of the family has suffered from a similar complaint. The significance of these preliminary observations and inquiries is of importance when considering the possible conditions that can account for the illness. For the sake of brevity I have set down in tabulated form the diseases, or rather some of them, which can occur at this age with splenic enlargement. They are (*i.*) syphilis, (*ii.*) tuberculosis, (*iii.*) congenital general œdema, (*iv.*) polycythæmia, primary or compensatory, (*v.*) anæmias, (*vi.*) congenital leuchæmia, (*vii.*) sepsis, (*viii.*) congenital atresia of the bile ducts, (*ix.*) congenital acholuric familial jaundice and (*x.*) Gaucher's disease.

It will be seen at once that some of these are exceedingly rare. Of the commoner diseases, my experience would lead me to place them in the following order: syphilis, anæmias of irregular form, tuberculosis, sepsis and congenital atresia of the bile ducts.

Syphilis.

Syphilis is the commonest cause of splenomegaly in the first six months of age. If detected in the newly born, the enlarged spleen is generally associated with a peculiar, pale, brownish-yellow discoloration of the skin and pemphigus or bleb-like lesions on the palms of the hands and soles of the feet. This syndrome, splenomegaly, pemphigus and *café au lait* colour and, one might add, slight enlargement of the liver and general flaccidity, is almost invariably fatal. Should such an infant live any length of time, ascites and jaundice occur. This type of congenital syphilis with splenomegaly is sharply marked off from the common form, in which the infant is born apparently healthy and shows no evidence of the disease till some weeks later, when snuffles, rash, wasting, suggestive facies, rhagades, dilated cranial veins, etc., occur and proclaim the diagnosis. At or about this time splenic enlargement becomes noticeable and competent observers variously estimate that it is present in 30% to 78% of all cases. There is still another clinical picture which is met but rarely in this age period and in which syphilitic cirrhosis of the liver is responsible for the symptoms and signs. The infant is atrophic with large, shining abdomen over which course large, superficial veins. The liver and spleen are enlarged. Jaundice is absent as a rule, but the infant is pale and wasted and the abdominal cavity contains much fluid. The outlook is bad and death occurs sooner or later, though it may be deferred considerably longer than in the first variety mentioned.

Tuberculosis.

Henoch has said that the commonest disease of the spleen in childhood is tuberculosis. That tuberculosis is a frequent cause of splenomegaly under six months of age is not generally known. Most patients with tuberculous meningitis in very early life show an enlarged, often tender spleen. Last week at the Children's Hospital Dr. Webster made a *post mortem* examination on such an infant, aged eleven weeks, the cause of death being general tuberculosis. The peculiar susceptibility young infants have towards the tubercle bacillus is exemplified in these early cases and there is no doubt whatever that quite a short exposure to a person afflicted with pulmonary tuberculosis is sufficient to inoculate a baby. The splenic enlargement may afford a clue to the diagnosis, even before definite symptoms indicating invasion of the meninges are detected.

Congenital General Œdema.

Congenital general œdema is a rare condition described by von Reuss and has been noted chiefly in premature infants. Œdema, a moderately enlarged spleen and a blood picture showing erythroblasts and myeloblasts are the essential features. Maternal toxins, not syphilitic, are possible factors in causation, as many are born of mothers with nephritis of pregnancy. An infant showing generalized hard œdema, especially of the lower limbs, enlarged spleen, pallor and a blood picture of one and a half million red blood corpuscles, with lymphocytes and poly-morpho-nuclear cells as fifty-eight is to thirty-six, came under notice at the Children's Hospital and in this infant Dr. Webster was able to demonstrate spirochætes in the liver, spleen and adrenals, as well as a specific osteo-chondritis.

Anæmia accompanied by splenomegaly is difficult of classification at this early age. Competent observers have despaired of describing any characteristic blood picture applicable to the varying forms of anæmia which occur in this post-fœtal period.

Anæmia and Congenital Leuchæmia.

Congenital leuchæmia is extremely rare and can be differentiated even at this age with some degree of accuracy by blood examination. Gordon Ward

quotes several cases from the literature and recently an instance of the spleno-medullary type was recorded. The disease is neither hereditary nor congenital. Recently a woman suffering from leuchæmia gave birth to twins whose blood pictures were normal. Von Jaksch's anæmia, on the other hand, may occur in twins and to that extent is familial.

Frequently one sees quite young infants with a moderate degree of anæmia and a slightly enlarged spleen, who are active and well in other respects. The ætiology is often quite obscure. One looks in these infants for malnutrition or disease on the part of the mother and excludes as far as possible syphilis, malaria, alcoholism and other intoxications, in many cases without result. Yet very many of these infants with pallor and mild splenomegaly do perfectly well without treatment of any kind.

Such an authority as Marfan believes that primary anæmias do not exist in nurslings and that the anæmia and enlarged spleen are due to some acute or chronic infection. If the percentage of myelocytes is unduly high, one may be suspicious of the later occurrence of a von Jaksch's anæmia. There is a disposition on the part of some authorities to give credence to the existence of an aplastic type of anæmia, due, it is thought, to a congenital want of development of the bone marrow (hypoplasia). As this is but rarely associated with splenic enlargement, it can be dismissed from consideration here. Undoubtedly a large percentage of children suffering from these so-called simple anæmias (even showing myelocytes in the film) with slight splenic enlargement recover without any treatment whatever. If, however, the splenic enlargement is very obvious, it is more likely to be a precursor of the von Jaksch's type, in which the prognosis is not so good.

Polycythæmia.

Polycythæmia with enlarged spleen occasionally, though rarely, persists as a continuation of the normal polycythæmia existing at birth. It would seem to be a counterpart in infancy of the acquired disease in adults known as Vaquez-Osler disease. This primary form is very rare. A secondary type, compensatory in origin, is met with in infants suffering from congenital heart malformation with enlarged spleen. It is also met in conditions reducing the oxygenating area in the lungs, as in emphysema, fibrosis, atelectasis, and occurs from over-stimulation of bone marrow by toxins of syphilis, tuberculosis, malaria, etc.. Patients with polycythæmia and full, red, dry face, cyanosis, peripheral congestion, swollen feet, epistaxis, dyspnœa and enlarged spleens are not often recognized as such till later childhood or even adult life, but the condition does occur early and should be identified.

Acute Infections: Sepsis.

Jaundice associated with an enlarged spleen may occur in an infant under six months of age in a variety of diseases. Syphilis we have already considered. Of the others, acute infections, congenital atresia of bile ducts, such familial diseases as familial hæmolytic splenomegaly or acholuric family jaundice and Gaucher's disease, though rare, require differentiation. Malaria is transmissible to the fœtus and paroxysms may occur within the first four or five weeks of life, with enlargement of spleen, fever and icterus. I mention this merely for the sake of completeness. If on or about the fifth day a baby developes fever and icterus and becomes progressively worse, with an enlarging liver and spleen and hæmorrhages, some acute septicæmic condition is probably present. Examination of the umbilicus, conjunctivæ, vulva, etc., may reveal the presence of sepsis. These were the cases so frequently seen years ago, but of rare occurrence in these times. The prognosis is not always bad, for on several occasions in my own experience recovery has ensued. It must not be assumed that sepsis is the only explanation of these conditions, for some have been definitely proved to be due to intestinal infections dependent on the *Bacillus coli*.

Congenital Atresia of the Bile Ducts.

In congenital atresia of the bile ducts the infant may be born jaundiced or icterus may occur within the first few weeks of life and rapidly increase in intensity. Bile pigment appears in the urine and the stools are acholic. Wasting sets in sooner or later and is progressive. The liver becomes very large and the spleen easily palpable. Hæmorrhages may be apparent in the skin or occur from the stomach, navel or bowel and may cause death. Thompson records one patient living ten months. Of four patients seen by me, one lived eight months.

Acholuric Hæmolytic Jaundice and Gaucher's Disease.

Acholuric hæmolytic jaundice or familial hæmolytic splenomegaly and Gaucher's disease are two rare affections of which mention might be made in this age period, though they may not become diagnosable till the child is considerably older. As Dr. Cowen's admirable paper has dealt with these *in extenso* to-night, my remarks will be brief and refer mainly to points of difference between them. There is a distinct family history traceable in both and in both enormous spleens are present. In the former jaundice of a hæmatogenous type is present; in the latter a brownish or greyish pigmentation, not exactly icteroid, affects the face, scleræ, neck and hands. Cirrhosis of the liver with enlargement characterizes Gaucher's disease, but does not occur in the hæmolytic variety, whilst increased fragility of the red corpuscles and anæmia are essential to hæmolytic splenomegaly, but do not occur in Gaucher's disease. Personally I have had no experience of Gaucher's disease, never to my knowledge having seen a case.

Grave Familial Jaundice.

Some years ago I saw a female baby, three days old, jaundiced and drowsy, who rapidly became worse and died the same day in convulsions. She was afebrile and the spleen was palpable, but not very readily. The two previous infants, both females, had died in a similar manner. I made a *post mortem* examination, but could find no definite cause of death. Subsequently a male child was born healthy and lived. I had before that met with a somewhat similar sequence in another family.

Quite recently Rolleston has published an account of this condition, which he calls grave familial jaundice of the new-born and favours maternal toxæmia as the cause.

Floating Spleen.

Before leaving this age period, one other condition requires mention, as it may be accidentally met with and prove puzzling. I refer to floating spleen. When this is felt in sites other than normal, the impression is apt to arise that the organ is enlarged. In *The Lancet* of March, 1921, Southam reported a case of a boy, aged six years, who was operated upon for an acute attack of pain and swelling in the right iliac fossa, which proved to be a spleen twisted two and a half times round its pedicle.

The Second Age Period.

Passing next to the age period between six months and four years, this will be familiar to you as the age when deficiency and nutritional diseases manifest themselves and the large group of gastro-intestinal affections are most in evidence. Many of the diseases which came under review in the previous group, are met with here. My remarks, however, will be confined mainly to splenic enlargement accompanying rickets and von Jaksch's anæmia.

Deficiency and Nutritional Diseases.

Rickets is the commonest of the so-called deficiency diseases, although not itself purely a deficiency disease, and is moderately prevalent in Victoria in mild degree. Splenic enlargement is found to be common enough when carefully sought, but whether it is related in some way to the anæmia which is one of the distinguishing features of rickets or whether other factors play a part in its production, is still the subject of controversy. The typical features of the rhachitic infant are well known. The large, square head, with delayed closure of the fontanelle, the small chest, showing the characteristic bony deformity, *e.g.*, rickety rosary, Harrison's groove, splayed lower ribs, etc., the protuberant abdomen, with *diastasis recti* and palpable spleen, the enlarged epiphyses of some of the long bones, the bent limbs, atonic musculature, defective teeth, pallor and sweating, make up a picture no one can forget. The disease as depicted here, though common enough in Great Britain and the older countries, is rarely shown to full perfection in this country. Indeed, in Queensland, the presence of even the milder varieties of rickets is denied. These rhachitic children are noted for their proneness to develope tuberculosis and consideration of the one should always cause due regard to be paid to the possible presence of the other. There is no clinical method of splenic examination which will enable us to differentiate the various diseases in which splenomegaly commonly occurs. It is thought that tuberculous and syphilitic spleens, when palpable, are firmer to the touch than those of most of the more acute infectious diseases, such as typhoid. Some are very tender and this applies particularly to the spleen of acute tuberculosis. The syphilitic spleen is, as a rule, less tender than most, but one child with unusual tenderness has revealed on *post mortem* examination extensive peri-splenitis. Some of the massive splenomegalies showed tenderness on handling (hæmolytic jaundice and leuchæmia), though, as a rule, it is my impression that large spleens do not appear to be very tender to ordinary palpation. It is interesting to note at this point that Moynihan considers that a spleen has doubled its size when it becomes palpable.

The ordinary gastro-enteric infections of infancy do not cause enlargement of the spleen. If, however, the disease has existed some time and induced an anæmic state, the spleen, as in rickets, appears to resume its fœtal function as a blood-forming organ as well as a destroyer of red corpuscles and becomes enlarged.

Von Jaksch's Anæmia.

As the end of the lactation period is the most favoured age for von Jaksch's anæmia to appear, a few words explanatory of this disease is desirable. It is a disease whose chief characteristics are anæmia of severe degree and splenomegaly, and its age of election from six months to two years. Horwitt, in the *Archives of Pediatrics* for November, 1921, says that generally there is a history of pallor and lowered vitality from birth, perhaps of prematurity or of twin birth. Often there is a history of early resort to artificial feeding, digestive disturbances and in some cases of acute infectious disease. Occasionally it appears to start in a child previously normal. Rickets and syphilis have been suggested as features in the ætiology. The disease developes insidiously, with pallor, anorexia and weakness. The pallor is of a waxy, yellow tinge, reminiscent of pernicious anæmia. The abdomen is protuberant, the spleen enlarged and often extending beyond the navel. It is smooth and movable, somewhat elastic to the touch, sometimes tender. The liver and lymph nodes may in some cases be very slightly enlarged. Hæmic murmurs are audible. Now and then ecchymoses are found, both into the skin and from mucous membranes. Blood examination reveals a severe anæmia The essential features are gross reduction in the red corpuscles and the presence of myelocytes and nucleated red corpuscles, often in considerable numbers. The white cell count is considerably increased and a lymphocytosis is generally noted. Blood features which in the adult are considered characteristic of leuchæmia or of pernicious anæmia, are here found side by side in the same case. Authorities are loath to regard this as a primary anæmia, because similar blood pictures have been found when the cause discovered *post mortem* was acute tuberculosis, gastro-enteritis, etc.. Kala-azar is said to present an almost identical blood picture in children, though a leucopenia is more often found than in von Jaksch's anæmia. The course is generally sub-acute, but occasionally a fatal issue may supervene in a few weeks. With remissions the disease may run a few years. The fatal termination is in a majority of cases due to intercurrent affections, notably bronchopneumonia. The mortality is variously estimated at 25% to 50%, the more favourable prognosis dis-

tinguishing this disease from leuchæmia, which is always fatal.

Leuchæmia.

One of the largest spleens met with in childhood is that of acute leuchæmia. For practical purposes the only form justifying consideration in this short paper is the acute lymphatic type. Whilst instances of the disease have been recorded as occurring at birth, a leuchæmic mother recently gave birth to twins, neither of whom showed any evidence of the disease. It occurs chiefly from seven months of age onwards, but inquiry from Dr. Webster and Dr. Graham at the pathological department of the Children's Hospital place the age of election between three and eight years. Its onset may be insidious, with anæmia, lassitude, adenopathy and splenomegaly rapidly increasing. Commonly, however, it commences suddenly, like an acute infectious disease, in the very young may be with a convulsion, and death generally takes place within two months. Should the child live longer, the possibility of the myelogenous or mixed types requires consideration. Bass, at Mount Sinai Hospital, New York, asserts that no affected child has ever been known to live over a year. One baby, aged seven months, was only ill four days when death occurred.

An acute onset with nervous symptoms may simulate acute cerebro-spinal meningitis with fever, convulsions, coma and hæmorrhages. At first the glandular enlargement may be only slight, cervical and associated with tonsillitis or stomatitis, but it rapidly increases and spreads to other regions. Pallor and enlargement of the spleen soon become prominent, later hæmorrhagic gums and a general tendency to hæmorrhages. The disease may assume a typhoidal character. The blood changes are characteristic. The white count increases enormously and more remarkable still is the transformation in the differential count, over 90% being large lymphocytes (so-called). The remainder are mainly poly-morpho-nuclear neutrophiles. The red cells and hæmoglobin are much reduced. The actual origin of the so-called large lymphocyte which dominates the blood picture in these cases, is the subject of much controversy. It is now regarded by many as medullary in origin (pre-myelocyte), so that the disease may even yet be termed acute myelogenous leuchæmia. The differential diagnosis between the two can only be made *post mortem* in many cases.

Dunn has succinctly summed up the clinical aspect when he says, if the physician is confronted with a disease in a child of acute febrile onset resembling that of an acute infection, especially if associated with stomatitis, tonsillitis and the rapid development of a tendency to hæmorrhages and splenic enlargement, he should remember the possibility of acute lymphatic leuchæmia. If the blood examination then shows, even without a marked increase in the white count, a lymphocytosis of 90% or over, a diagnosis of acute leuchæmia can be made. They die from exhaustion, hæmorrhages, sudden heart failure and in one case at the Children's Hospital from œdema of the glottis as part of a general water-logging.

The Third Age Period.

The last period, from four years to puberty, is the school age, familiar to all as the age of acute infectious diseases. Any acute infectious disease may be accompanied by an enlargement of the spleen at some period of the disease.

Acute Infectious Diseases.

Certain acute infections, however, have splenic enlargement as a more or less constant accompaniment. They are typhoid fever, acute tuberculosis, septicæmia and ulcerative endocarditis. One has noticed also the frequency with which severe forms of acute diseases present an easily palpable spleen. Pneumonia, also diphtheria, especially if the child be under three or four years of age, are two which come readily to my recollection. In countries bordering on the tropics the possibility of malaria and kala-azar should always be borne in mind. No case of malaria developing in this State has come under notice, but I have seen patients from Queensland and the Malay States exhibiting moderate splenomegaly. Anchylostomiasis or hookworm disease, also active in Queensland, occasionally has some enlargement of the spleen. I have seen no instance of this in Victoria, but opportunity was afforded members of the last Congress in Queensland of seeing several patients whose chief symptoms were langour, mental dulness, anæmia, palpable spleen in some cases and œdema of extremities. The presence of the anchylostomum and ova in the stools were diagnostic.

Chronic Infections.

Of the more chronic and sub-acute conditions inducing splenic enlargement, attention should be directed to certain cardiac affections. In these consideration must be given to the effects of emboli, infarction and passive congestion in relation to the splenomegaly, also hyperplasia of the spleen as a feature in sub-acute bacterial endocarditis.

Syphilis in its more chronic form is common at this age and the splenomegaly, in common with anæmia, is often a source of much confusion in the diagnosis of splenic anæmia or Banti's disease.

Where chronic sepsis or tuberculosis with discharging wounds occurs alone or in a patient the subject of congenital syphilis, the presence of an enlarged spleen should at once raise the question of lardaceous or amyloid disease. Some years ago at the Children's Hospital lardaceous disease of the viscera was a well known and frequent exhibit in the *post mortem* room. Now it is rarely seen, a circumstance traceable, perhaps, to improvements in the treatment of chronic bone disease and congenital syphilis and possibly to a diminution in the incidence of the latter. The last possibility is my personal opinion only.

Still's Disease.

As Still's disease or chronic infectious arthritis of children is generally accompanied by splenic enlargement, brief mention of the symptomatology will be made. It is a poly-arthritis which most often occurs during this age period, though I have seen it begin as early as seventeen months. The

onset may be acute or insidious, with stiffness and swelling of the joints, especially of the hands and feet, though larger joints soon become involved. The affected joints tend to become fusiform in shape and rigid. Fever of an irregular type is an associated feature and wasting of muscles and secondary anæmia soon appear. The most distinctive feature next to the arthritic changes is the general enlargement of the lymph glands. There are periods of improvement, but the general tendency is towards chronic invalidity from ankylosis and death from intercurrent disease.

Hanot's Biliary Cirrhosis.

In Hanot's biliary cirrhosis, which is said to occur between five and fifteen years of age, the leading features are grossly enlarged liver, splenomegaly and jaundice. Crises of abdominal pain and pyrexia hasten the downward course, which is quicker the younger the child, death occurring in two or three months. A doubtful case in a child considerably under five years of age is the only instance I can remember of anything suggesting Hanot's disease.

Anæmia Gravis.

Pernicious anæmia as known in adult life is said not to occur under puberty. Odd cases, however, have been reported from time to time. Dr. Webster and Dr. Graham, in their clinico-pathological work at the Children's Hospital, confirm my experience in never having met with a typical case. A disease, however, termed *anæmia gravis* has been described (*The British Journal of Children's Diseases*, Volume XIX., 1922) by Poynton, Thursfield and Paterson, in which the essential features are severe anæmia, enlarged spleen, hæmorrhages and transparent pallor of insidious onset in children about eleven years old who generally die. The anæmia is not leuchæmia, nor a purpura, is not related to Hodgkin's disease and has many points of difference from pernicious anæmia of adults. The bone marrow is white. The authors seem to regard it as a possible aplastic type of anæmia occurring in children, but not in infants.

Hodgkin's Disease.

Of the remaining diseases with splenomegaly which occur between four years and puberty, only two of any importance remain, *viz.*, Hodgkin's and Banti's disease.

Hodgkin's disease, formerly synonymous with lymph-adenoma, but now maintained by many to be an infective poly-adenitis, is rare under four years of age. After this age it is much more frequent and several cases are exhibited every year at the Melbourne Pædiatric Society's meetings. The children are brought, as a rule, on account of a visible enlargement of a group of glands, such as cervical, axillary or inguinal. More rarely they are presented because of the pressure effects occasioned by mediastinal and retro-peritoneal growths. There is generally progressive anæmia of a secondary kind with leucocytosis and mild fever sometimes assuming a rather characteristic intermittent type and recurring at intervals. The spleen is usually easily palpable at some period of the disease. The glands have a tendency to vary in size and are generally discrete, though in massed formation, differing in this respect from tuberculous adenopathy, in which matting together is the rule. They simulate syphilitic adenopathy in the young, especially the acquired form. Secondary effects from pressure, such as dyspnœa, cyanosis, œdema and ascites, are evident in the later stages. The blood shows no characteristic changes, but Bunting emphasizes the value of an absolute increase in transitional cells as the distinguishing feature of Hodgkin's disease.

In differentiating leuchæmia, Hodgkin's disease and tuberculous adenopathy, the blood examination will exclude leuchæmia. A lymphocytosis pertains in the early stages of both Hodgkin's disease and tuberculosis and here Bunting's transitional cells may be helpful. Later in the disease a high polynucleosis would favour Hodgkin's disease rather than tuberculosis. Removal of a gland for histological report frequently clinches the issue, though I well remember one instance in which sections from different portions of the glandular mass showed the pathological structure of Hodgkin's disease, tuberculosis and lympho-sarcoma. The course of the disease is very chronic, but the prognosis is unfavourable. Sooner or later the child succumbs from pressure effects, exhaustion or intercurrent disease. Complete surgical removal of the glands in the early stages may materially alter the prognosis for the better.

Banti's Disease.

Banti's disease or splenic anæmia is rare in children. Isolated cases come under review from time to time and it is necessary to recognize it early, as it is one of the few diseases for which splenectomy provides the best treatment. Bartlett, in the *American Journal of Children's Diseases*, April, 1922, has collected the records of fifty-one cases of splenectomy in children under fourteen years of age and states that hæmolytic jaundice, Banti's disease, Gaucher's disease and von Jaksch's anæmia are conditions in children for which relief or cure by splenectomy may be the only treatment. Banti's disease occurring in the young is often confounded with von Jaksch's anæmia; in fact, splenic anæmia is a term that has been and still is applied, though wrongly, to both. In Banti's disease, however, splenomegaly is the primary lesion, unaccompanied, perhaps, by any other signs or symptoms. Later the blood shows the characters of a secondary anæmia, with diminution of red blood corpuscles, low colour index and either no appreciable change in the white cells or else a leucopenia with relative lymphocytosis. Corpuscular fragility is generally below normal and a feature of importance in diagnosis. Slight enlargement of the liver without jaundice may be noticeable. In the second stage jaundice is the big feature, whilst gastro-intestinal symptoms, loss of weight and cachexia are frequent accompaniments. In the third stage ascites sequential to hepatic cirrhosis occurs and hæmorrhages from the gastro-intestinal tract may be pronounced and cause death. Banti's disease is said to occur occasionally in families. The essential feature is that the splenic enlarge-

ment should precede the anæmia and hepatic cirrhosis, a point impossible to decide in many cases.

Neoplasms.

Neoplasms of the spleen, such as sarcoma, primary or metastatic, must be extremely uncommon in children. I cannot recollect having seen a single case of either and a careful scrutiny of the hospital records for the past eight years showed none. A search of the records for the past twenty years failed to reveal any instance of hydatid of the spleen.

Conclusion.

To conclude, I make no apology for the somewhat kaleidoscopic character of this paper; the very title, for which I am not responsible, would indicate that it cannot be otherwise.

My thanks are due to Dr. S. Patterson, of the Walter and Eliza Hall Institute, for his kindly preparation of the lantern slides, and to Dr. Webster and Dr. Graham, of the Pathological Department, Children's Hospital, for much assistance from their laboratory.

BILE PIGMENTS IN THE BLOOD.[1]

BY MAUD COWEN, B.Sc.,
AND
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THE normal process of blood destruction in the body is not fully known, but experimentally it has been shown to be a function of endothelial cells, *e.g.*, of spleen, or of serous cavities and of the Küpfer cells of the liver. Independently of the liver, hæmoglobin may be broken down to bile pigment in extravasations of blood. This has been shown experimentally in animals. The spleen and liver are usually considered to be the organs normally responsible for destroying effete red blood corpuscles.

In considering the general pathology of blood destruction we may regard the normal process as either in abeyance (this may be one factor in the causation of polycythæmia) or in excess, as in splenic anæmias. Secondly, hæmolysis may be caused by chemical, bacterial or animal toxins entering the body from outside or by toxins in the body, as in paroxysmal hæmoglobinuria. Thirdly, the sensitiveness of the corpuscles may be affected, especially in regard to increased fragility, as in acholuric jaundice.

The diazo reagent of Ehrlich has been shown by Van den Bergh[(1)] to give its characteristic bluish-violet colour with bilirubin. It may be used, therefore, as a test for bilirubin in the blood. Van den Bergh describes two main types of reaction in serum. The first, when serum and reagent mixed together give the colour, he calls the direct reaction. When the colour with the reagent is not given at once by the serum, but only when the proteins are precipitated from the serum by alcohol and the supernatant fluid added to the reagent after centrifuging, this is the second or indirect reaction. The direct reaction is described by Van den Bergh as occurring when there is jaundice due to obstruction in the biliary passages and the indirect reaction when due to functional defect in the liver cells. He describes a biphasic reaction (a reddish colour in the pure serum which slowly deepens to violet) as indicating that the jaundice is due both to func-

TABLE I..

No.	Provisional Diagnosis.	Final Diagnosis.	Jaundice.	Direct Test.	Indirect Test.	Quantitative Test.
1	Obstructive jaundice	Carcinoma of pancreas	+	+	+	5.4 units
2	Biliary colic	Gastric ulcer	0	—	+	<
3	Cholelithiasis	Adhesions	0	—	+	<
4	Obstructive jaundice	Cholelithiasis	+	+	+	12.1 units
5	Cholelithiasis	Cholelithiasis	+	+	+	10.0 units
6	Biliary colic	Salpingitis	0	—	+	<
7	Biliary colic	Cholelithiasis	0	—	+	<
8	Cholangitis	Hydatid of liver	+	+	+	3.35 units
9	Carcinoma of pylorus	Cholelithiasis	+	+	+	2.66 units
10	? Cholelithiasis	? Cholelithiasis	0	—	+	1.8 units
11	Malignant disease of abdomen	Carcinoma of stomach	0	+[3]	+	2.6 units
12	Biliary colic	? Obstructive jaundice	+	+	+	3.9 units
13	Biliary colic	Adhesions	0	—	+	<
14	Biliary colic	? Cholelithiasis	+	—	+	5.1 units
15	Biliary colic	Biliary colic	+	+	+	1.3 units
16	?	Secondary malignant disease of liver	0	—	+	1.1 units
17	?	Actinomycosis of liver	0	+[3]		?
18	?	Hydatid of liver	0	—	+	<
19	?	Adrenal tumours	0	—	+	<
20	Biliary colic	Cholelithiasis	0	—	+	<
21	Stone in common duct	Catarrhal jaundice	+	—	+	<
22	Catarrhal jaundice	Catarrhal jaundice	+	—	+	3.0 units
23	? Stone in common duct	?	+	+	+	10.0 units
24	Cirrhosis of liver	Cirrhosis of liver	0	+[3]	+	2.3 units
25	Jaundice	Cholelithiasis	+	+	+	8.6 units

[1] Read at a meeting of the Victorian Branch of the British Medical Association on October 4, 1922.
[2] Biphasic. [3] Hæmolysed serum. < = Less than could be estimated colorimetrically.

TABLE II..

No.	Provisional Diagnosis.	Final Diagnosis.	Jaundice.	Direct Test.	Indirect Test.	Quantitative Test.
1	Pernicious anæmia	Pernicious anæmia	0	—	+	<
2	Pernicious anæmia	Hæmorrhoids; anæmia	0	—	+	1.1 units
3	Pernicious anæmia	Pernicious anæmia	0	—	+	2.0 units
4	Familial acholuric jaundice	Familial acholuric jaundice	0	—	+	2.0 units
5	Pernicious anæmia	Splenic anæmia	0	—	+	2.1 units
6	Anæmia	Pernicious anæmia	0	—	+	2.0 units
7	Acholuric jaundice	Acholuric jaundice	0	—	+	0.88 units

tional alteration of the liver cells and to obstruction in the bile passages. McNee[2] has drawn attention to the use of this test in differentiating obstructive from other types of jaundice.

During the past few months we have carried out the test on the serum of a series of patients. The results are shown in the accompanying tables. Table I. deals with patients with obvious or suspected hepatic disease and Table II. with patients suffering from anæmia.

The inferences to be drawn from these figures are:

(*i.*) We have in the Van den Bergh test a delicate reaction for bile pigments in the serum. We find it more sensitive to bile pigment than Gmelin's test, for instance.

(*ii.*) Bilirubin is usually present in appreciable quantities in the blood.

(*iii.*) Jaundice in the patient and a direct reaction with Van den Bergh's test were found together in all instances but five. One of these patients (Case 11) was not clinically jaundiced at the time of testing, but stated that he had been jaundiced some time previously; his serum gave the biphasic reaction and quantitatively an increased amount of bilirubin was present; at operation a pyloric carcinoma with secondary deposit in the liver was found. In Case 24 (cirrhosis of liver) the patient was not jaundiced and the reaction was biphasic. These cases are compatible with Van den Bergh's explanation of the biphasic reaction occurring when there is combined obstruction of ducts and functional alteration of the liver cells. Two patients (Cases 21 and 22) suffered from catarrhal jaundice and, although jaundiced, gave no direct reaction in the serum, pointing to a functional alteration of liver cells rather than an obstruction in this disease. Another patient (Case 14) was admitted to hospital with symptoms of biliary colic; he was jaundiced, but his serum did not give the direct reaction. He refused operation and the final diagnosis was not completed, but the result of the test would lead us to assume a defect of function rather than an obstruction.

These results are suggestive only, but they are published to incite others to make trial of a test for liver function, which appears likely to be of considerable use in an avowedly difficult field.

References.

(1) Van den Bergh: *La Presse Médicale*, 1921, page 441.
(2) McNee: *The British Medical Journal*, 1922, Volume I., pages 716 and 783.

< = Less than could be estimated colorimetrically.

FRACTURES.[1]

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WHEN you did me the honour of asking me to read a short paper at your annual meeting to-day and your Secretary suggested "Fractures" as the theme, I was delighted to embrace the opportunity of renewing old acquaintances in the West and receiving the benefit of your experience. However, the subject is a difficult one in that finality has not been reached in the treatment of bone injuries and you are all well versed in the methods adopted during the great war. One cannot help mentally comparing the equipment supplied for treating fractures during the Gallipoli campaign with that in use in the later stages of the war. In civil hospitals a few years ago in Sydney the surgeon regarded the admission of a fracture patient into one of his beds as a misfortune. To-day his interest is aroused and he realizes the importance of securing good results. The application of X-rays to the diagnosis of fracture of bone has contributed much toward an accurate interpretation of the physical signs of fracture. The routine use of general anæsthesia in the examination and initial treatment has saved the patient much pain and made the application of splints more accurate.

The use of splints and apparatus generally which allows the patient a degree of mobility in bed, has greatly added to his comfort and that of his nurses. At the Sydney Hospital the employment of a modified Balkan frame, Hodgen's and Thomas's splints and a body sling by which the patient is able to raise himself off the bed, if necessary, has robbed fractures of the lower extremity of most of their discomforts and the patients are most contented in the fracture wards.

Speaking generally of the treatment of fractures, it cannot be too strongly emphasized that very careful examination must be made, not only of the obviously injured part, but of the patient himself. Accurate notes and measurements must be made immediately and during the period of treatment. The near relatives should be informed of the serious nature of the injury, the complications that may arise and a guarded prognosis given. The possibility of severe internal injury and of constitutional disease must not be lost sight of; the latter may be

[1] Read at a meeting of the Western Medical Association at Wellington, New South Wales, on September 22, 1922.

the primary cause of the fracture. The *Workmen's Compensation Act* has always to be reckoned with and our statements must be well considered.

The success of any form of treatment depends largely on the constant personal care of the medical attendant. A change in the nursing staff may easily upset his work, especially when overhead gear with weights and pulleys and extension applied directly to the bone is being employed. Bright surroundings are helpful and the possibility of getting out in the sunshine is often no small factor in the treatment.

The initial treatment often profoundly influences the final result. The sooner the injured limb is attended to the better.

The question of transportation is important; the less, the better for a fracture. In every case traction should be applied to avoid laceration of muscles, vessels and nerves by the sharp fragments and to prevent over-riding and stripping of the periosteum. The apparatus should be light, strong and easily applied. The Thomas's knee splint is invaluable and easily made by a blacksmith. The splint should fit the patient; no endeavour should be made to make the patient fit the splint!

The functional result obtained is the most important thing. It is not advisable for the patient to see X-ray plates taken after a good functional result has been secured, as he and his friends are often disappointed. However, X-ray plates are a useful record to keep. Courts of law are apt to regard it as an act of negligence on the part of the medical attendant if the opportunity of having an X-ray examination made has not been grasped. In the city many bad compound fractures are met with as the result of tram, motor-car and wharf accidents. The best treatment to be adopted and the question of amputation or the employment of conservative measures cause great anxiety even to the most experienced surgeons.

The injury to nerves and vessels and the amount of damage to the skin have to be carefully weighed. The history of the accident is often a great help in coming to a decision. For example, in machinery and tram accidents much more damage has been done to the skin than is immediately apparent. The stretching and bruising of the skin, with consequent lowering of its vitality for a considerable area beyond the obvious site of injury, is to be expected. Another factor is that wounds contaminated with street dirt often contain virulent organisms and in such cases the immediate injection of anti-tetanic serum is indicated. On the other hand, my experience is that the oil and grease on the limbs of those working in engineering workshops do not prevent healing by first intention. My practice in hospital in dealing with compound fractures is to prepare myself as for any surgical operation and then put on a second pair of gloves, the latter to be removed later on by a nurse. I find it best to clean up the surrounding skin and wound myself. Hair is dry shaved, ether is employed to cleanse the skin, always working away from the wound, and then tincture of iodine applied. Next the wound itself is cleansed by mechanical removal of all obvious dirt or foreign body by means of dry sterile gauze or gauze moistened with one in forty carbolic acid. With forceps and scissors the edge of the damaged skin is cut away. The outer gloves are removed and my assistant places sterile towels around the operation area.

If possible, a tourniquet is not applied, as I am sure it has a bad effect on the tissues and favours the growth of organisms. If a tourniquet has been applied before admission to hospital, it is removed at once and bleeding points caught with forceps, if possible; these are left on until the arrival of the patient in the operating theatre. Loose fragments of bone are not removed if it is at all possible to place them in position; damaged muscle is cut away and the wound left open or the wound held together by a few loosely applied silkworm gut sutures.

Drainage is amply provided for by free incision, if necessary. Drainage tubes are avoided. If the wound is very dirty, Carrel-Dakin tubes are placed in position and held there by gauze loosely packed in. When the Carrel-Dakin method is not applied and there is a raw surface, a thin layer of gauze soaked in sterile liquid paraffin is placed next to the wound and gauze moistened with a mild antiseptic fluid placed outside.

The splint used is such that the wound can be dressed without the limb being disturbed. The wound is of primary importance, the fracture secondary.

After a few days exposure of the wound to the direct sunlight is practised, if possible. The use of peroxide of hydrogen in any but superficial wounds is not advisable, as the gas formed tends to open up the tissues and perhaps new avenues for infection. During the year we have had several cases of gas gangrene occurring in compound fractures in patients admitted a considerable number of times after the accident.

In fractures of the lower extremity the prevention of foot-drop in both the injured and uninjured limb is of vital importance. Constant care has to be exercised to combat wasting of the *quadriceps extensor* muscles and consequent weakness of the knee joint in walking.

In the case of the upper extremity the preservation of the movements of the joints and especially of pronation and supination of the forearm and dorsi-flexion of the wrist must be secured.

Open operation is not employed nearly so often as a few years ago and conservative measures are more favoured. The principles laid down by H. O. Thomas and Sir Robert Jones may be reiterated:

(1) A fracture is a potential deformity and must be regarded as such.

(2) Strong, steady, fixed extension with corresponding counter extension is required, as distinguished from forcible, intermittent pull, succeeded by a period of muscular relaxation.

(3) Correct alignment in conjunction with obliteration of shortening must be attained.

(4) Circular compression must be avoided, as it tends to produce ischæmia.

(5) Union may be delayed. This is sometimes unavoidable, but there is a vast difference between delayed union and non-union.

(6) Union of a fracture does not imply its consolidation.

Before referring briefly to some individual fractures, may I urge the necessity of a regular examination of the patient apart from the injured limb during treatment. If confined to bed, the chest should be examined and also the urine; pyelitis and cystitis are not uncommon. Bed sores easily develope and the medical attendant will do well occasionally to see for himself that all is well with the patient's skin.

The Skull.

Fracture of the Vault.

If in doubt, turn down a flap of scalp and examine the skull. If there is a depression or a linear fracture with signs of hæmorrhage I always trephine.

Fracture of the Base.

My experience is that, except in special cases, operation is not to be advised. If one pupil is dilated and fixed and operation is decided upon, the surgeon should decompress on the side on which the pupil is dilated.

Fractures of the Bones of the Face.

Depression of the malar bone can usually be relieved by immediate elevation by means of a small periosteal elevator inserted through a small, transverse skin incision below the bone.

Fractures of mandible are best treated by dental splint, but if teeth are present, much can be done in the absence of a dental surgeon by wiring the teeth together with silver wire.

The Chest.

Fractured ribs with lung injury may give rise to surgical emphysema, which usually subsides and may sometimes be relieved by suitable bandaging. I once saw a patient literally blow his skin up with air after such an injury. Air could be felt beneath the skin from his head to his feet. The neck was so swollen that he was almost suffocated. The treatment employed was an incision over the damaged rib and the insertion of a large rubber tube, the distal end of which was placed in a bucket of water on the floor. Air was forced through the tube with expiration, but was not sucked in during inspiration. The emphysema was relieved immediately.

For fractures of the clavicle I must confess that I like Sayres's method of using broad adhesive plaster to fix the shoulder. Some surgeons condemn the method.

The Upper Extremity.

Fractures of the head and neck of the humerus are treated by abduction and extension.

Fractures of the shaft of the humerus are treated by Robert Jones's humeral traction splint. This may be used for fractures of the forearm also, as supination and extension of the forearm can be secured as well as of the humerus.

Fractures in the region of the elbow joint, except of the olecranon, are put up after adequate and complete reduction in the fully flexed position. Great care must be taken that swelling does not cause pressure on the vessels; constant observation is required.

For Colles's fracture complete reduction is the essential part of treatment. This can only be done under anæsthesia. The splint must keep the fragments in position and the hand in ulnar deviation. Early movements of the fingers should be encouraged.

The Pelvis.

In fractures of the pelvis the surgeon should examine the patient for other serious injury; the bladder is especially liable to be involved. In most cases a firm bandage and sandbags supply all that is necessary.

Some years ago a patient was under my care over whose pelvis the wheel of a loaded cart had passed. Several bones of his pelvis were broken and blood issued from his urethra. A catheter was passed and apparently entered the bladder, but only blood was obtained. Taking the case to be one of ruptured urethra, I opened the bladder suprapubically and was surprised to find that it contain clear urine and no blood. The catheter was lying in the space of Retzius and the urethra had been torn completely away from the bladder. With difficulty retrograde catheterization was performed and after many tribulations the patient made a good recovery.

The Lower Extremity.

Accurate measurements of both legs are necessary in all cases of fracture of the lower extremity. The measurements should include that from the anterior superior iliac spine to the tip of the medial malleolus and the distal edge of the patella should be located. Flexion of the hip and knee is to be desired and owing to the pull of the muscles extension must be used. This may be secured indirectly by means of adhesive plaster or Sinclair's glue, which consists of fifty parts of glue, fifty parts of water, two of glycerine, one of calcium chloride and one of thymol.

In fractures of the head and neck of the femur a certain amount of abduction is necessary. Either a Thomas's splint or a Hodgen's splint is used. Both are excellent, but Thomas's requires careful fitting and constant care in its adjustment. At the Sydney Hospital Hodgen's splint is most favoured.

In fractures of the shaft and in supracondylar fractures with much overlapping the Schmerz hook is most valuable, as a direct pull is obtained on the bone and shortening is easily overcome and the patient is very comfortable. In compound fractures it is very useful. Of course, it must be remembered that the patient's friends are apt to regard it as a crude method. The main points in its application are:

(1) It should be applied under general anæsthesia for preference.

(2) It should grip the femur above the condyles firmly and should ride on the hard cortical layer of bone without moving over it or penetrating it. It should not sink through into the cancellous tissue.

(3) The skin should be sterilized and pulled upwards as far as possible before the points are in-

serted, so that when the shortening of the limb is overcome, the skin is not dragged upon.

(4) The hook is left in position for two to three weeks and then replaced by ordinary strapping extension.

A smaller variety of hook may be used in fractures of the lower third of the tibia and in compound or very bad Pott's fracture.

Pott's fracture may be regarded as an outward and backward dislocation of the foot and this must be completely corrected. After union has occurred and is firm in fractures of the middle and lower thirds of both bones and in Pott's fracture, the patient should be allowed to walk with a light outside iron extending from the heel of the boot to five centimetres below the knee joint and the inner side of the sole and heel of the boot built up one centimetre to prevent valgoid deformity.

All patients with fractured femora should wear a walking calliper for at least eight weeks after union of the bone is considered to be sound enough to permit the patient to walk without risk. The walking frame is much better than crutches for the patient on getting on his feet again after being confined to bed.

The following measurements are required in ordering walking calliper splints:

(1) The circumference of the thigh opposite the perineum (A).

(2) An oblique measurement taken by passing the tape round the top of the thigh below the tuberosity of the ischium and above the great trochanter, the tape being drawn tight (B).

(3) The vertical distance from the tuberosity of the ischium to the under surface of the heel (C).

Remarks should be made upon any peculiarity of the shape of the limb.

All measurements should be actual upon the patient, no allowance being made for clothing or boots.

In conclusion may I thank you for the patient way in which you have listened to what has been, I fear, a rather disjointed paper. It is really only possible to appreciate the treatment of fractures in a fracture ward, where the patient and the apparatus in use can be seen.

Reports of Cases.

A COMPLETE CASE OF LEUCHÆMIA IN CHILDHOOD.

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Clinical History.

A GIRL, aged five years and six months, was admitted to the Melbourne Children's Hospital on August 16, 1920, in a serious condition with grave anæmia and made a rapid and apparently complete recovery. She was readmitted, however, on December 17, 1920, with acute leuchæmia and died on February 5, 1921.

The child had been languid and easily fatigued for about a year before admission and it had been noticed that her skin was frequently affected by sores which did not easily heal. For a fortnight she had had pertussis and her general condition had become much worse than usual. For a week she had been in bed, had developed a severe degree of anæmia, her cervical glands had become very swollen and she had been distressed with coughing and expectoration. For three days a bruise had been present over her left eyebrow and a purpuric rash on her neck and the front of her body and a trophic sore had appeared in the sacral area. Her breath had been foul for two days and on the night before admission her respirations had become rapid and her temperature was 38.3° C. (101° F.). Her appetite had remained fairly good, she had not vomited and defæcation and micturition had not been noticeably abnormal. In the past she had had measles and on several occasions had complained of sore throat. The family was a healthy one.

On August 16, 1920, the child was apathetic, her skin and gums were white and, in addition to the external features noted above, a hæmorrhagic herpetic eruption was present on the lips and also an abrasion on the left leg. She had an extensive left-sided pneumonia, her limbs were somewhat spastic, her urine reacted feebly to the Rothera test for the presence of acetone bodies; no enlargement of the spleen was detected and the throat swab taken in a routine way on admission resulted in the growth of coccal forms only.

The results of examination of her blood showed the hæmoglobin value to be 20% (Sahli), the red blood corpuscles to number 800,000 and the leucocytes 6,800 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 9%, eosinophiles 1%, large lymphocytes 8% and small lymphocytes 82%. In the blood film no nucleated red blood corpuscles were observed, but gross anisocytosis and some poikilocytosis were noted. Hæmoculture resulted in a growth of *Staphylococcus pyogenes albus* after sixty hours, perhaps from skin contamination.

On August 17, 1920, she received four hundred and fifty cubic centimetres of blood from an unrelated, healthy, compatible donor. Transfusion was performed intravenously by the indirect citrate method under local anæsthesia. No rigor or elevation of temperature occurred, but her cheeks became pink and next day a considerable improvement in her general condition was noted. However, on the second day a severe stomatitis was present, with bleeding from the lower lip and gums, her whooping cough became exhausting, she had a profuse, fœtid, muco-purulent, nasal discharge and her general condition was not good. Though nothing of a membranous nature was present, nasal and throat swab cultures were prepared and each of them resulted in the growth of coccal forms.

On August 21, 1920, a second transfusion was made, similar in all respects to the preceding one. She had a rigor and elevation of temperature to 40° C., but on the following day great improvement manifested itself.

On August 26, 1920, the Klebs-Löffler bacillus was reported to be present in the culture of a swab from her throat. A small dose of diphtheria antitoxin was administered subcutaneously and the administration of anti-pertussis vaccine was commenced.

On August 27, 1920, an acute parotitis required fomentation; it subsided in a few days.

On September 2, 1920, the Klebs-Löffler bacillus was again found in a throat swab culture, but no similar organisms were grown from the subsequent swabbings.

By September 3, 1920, she was quite comfortable and on September 10 the red cells numbered 4,350,000 per cubic millimetre, with a hæmoglobin value of 94%.

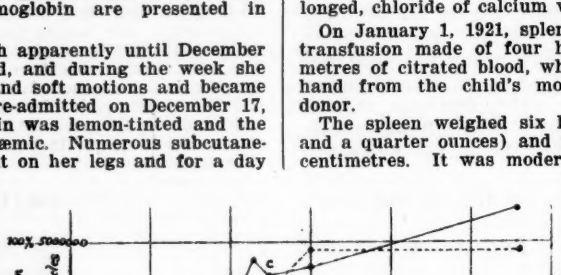
On September 15, 1920, the red cells numbered 4,800,000 and the white cells 4,000 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 63%, basophile cells 1%, large lymphocytes 25%, small lymphocytes 6% and myeloblasts 5%.

She appeared to be in perfect health, her temperature had not been above normal from September 1, she was taking full diet eagerly, her convalescence had been uneventful and on September 16, 1920, she was discharged from hospital.

On October 11, 1920, she was brought to the out-patient department by appointment and seemed to be well. Blood examination was made and showed the hæmoglobin value to be 104% (Sahli), the red cells 4,850,000 and the white cells 9,600 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 48%, eosinophiles 3%, large lymphocytes 16%, small lymphocytes 22% and "large mononuclear cells" 11%.

The progressive red blood corpuscle counts and estimations of percentage of hæmoglobin are presented in Graph I..

She remained in good health apparently until December 10, when diarrhœa commenced, and during the week she passed frequent tarry, small and soft motions and became pale and languid. She was re-admitted on December 17, 1920, and on this day her skin was lemon-tinted and the mucous membranes looked anæmic. Numerous subcutaneous hæmorrhages were present on her legs and for a day a petechial rash had been scattered over the front of her body. Her baby brother had pertussis and the patient had commenced to have paroxysmal coughing again, but she had not vomited and her appetite had remained good. Her spleen was greatly enlarged and occupied "two-thirds" of the abdomen. The lower border of the liver was two centimetres below the costal margin. No free fluid was detected in the abdomen. The cardiac dulness was estimated to extend one and a half centimetres to the right of the sternum and the apex beat was in its normal position. An apical systolic murmur, blurring of the heart sounds in the pulmonary area and a few scattered rhonchi were found on auscultation. Temperature and the respiratory rate were normal, but the pulse rate was one hundred. The urine contained acetone bodies in slight amount, though it was acid in reaction to litmus. No inflammatory appearances were seen on examination of the nose and throat and swab cultures resulted in the growth of coccal forms.



GRAPH I..

In the abscissa each division represents 100,000 red blood corpuscles and 20% hæmoglobin. In the ordinate each division represents ten days.

At the point indicated by an arrow and the letter *A* the red blood corpuscles numbered only 800,000 per cubic millimetre and the hæmoglobin 20%. The child was waxen in appearance and looked as if about to die. She was given four hundred and fifty cubic centimetres of blood of an unrelated donor.

At the point indicated by an arrow and the letter *B* there was an astonishing improvement in the appearance of the child. She was again given four hundred and fifty cubic centimetres of blood from the same donor. The improvement was accentuated and sustained; at *C* the child appeared to be restored to normal health, though the red blood cell count and the hæmoglobin percentage continued to rise. Several months later the child again became anæmic and continued observation (see Graph II.) showed the transition to an acute leuchæmia with a fatal termination.

Examination of the blood showed the hæmoglobin value to be 48%, the red cells to number 2,300,000 and the white cells 12,000 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 15%, large lymphocytes 40%, small lymphocytes 30%, large mononuclear cells 5% and myeloblasts 10%.

An occasional megaloblast was present in the film, but no other conspicuous changes in the red blood corpuscles were observed.

On December 28, 1920, the hæmoglobin value was 40%, the red cells numbered 2,000,000 and the white cells 9,000 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 11%, large lymphocytes 34%, small lymphocytes 33%, large mononuclear cells 6% and myeloblasts 16%.

Gross anisocytosis with presence of many macrocytes and frequent megaloblasts were features of the blood film.

On the preceding day she had passed two tarry stools and, in view of the facts that the diagnosis of acute leuchæmia, with its desperately bad prognosis, could now be made and that no known method of treatment essentially affects the prognosis and that she was steadily losing ground, it was decided that her spleen should be removed at an early date and that this procedure should be combined with X-ray therapeutic measures.

For ten days her temperature had been normal in the mornings and in the neighbourhood of 38° C. (100° F.) in the evenings and her pulse rate had averaged 135 pulsations in each minute. She had been taking an iron and arsenic mixture and as the blood coagulation time was prolonged, chloride of calcium was administered by mouth.

On January 1, 1921, splenectomy was performed and a transfusion made of four hundred and fifty cubic centimetres of citrated blood, which had been obtained beforehand from the child's mother, who was a compatible donor.

The spleen weighed six hundred grammes (twenty-one and a quarter ounces) and its long axis measured twenty centimetres. It was moderately firm in consistence and its capsule was perfectly smooth.

Microscopic section showed no increase in thickness of the capsule or of the stroma and no endothelial proliferation and the Malpighian corpuscles were of normal appearances. The spleen pulp contained fairly numerous cells of the large lymphocyte type (small rounded nucleus, moderate amount of clear protoplasm, staining evenly with eosin). The peripheral portion of a gland removed from the neighbourhood of the spleen hilus was of normal appearance microscopically, but the medullary area was obviously less cellular and examination under the high power disclosed the presence of giant cells strongly resembling megakaryocytes (large cells with embryonic, polymorphous nuclei and strongly eosinophile protoplasm) and large mononuclear cells similar to those in the spleen. There were also a few large cells with clear protoplasm and indented nuclei. The sinuses did not contain blood. It was considered that myelogenous tissue was present in a lymphatic gland and that the gland was not a hæmolymph gland.

On January 2, 1921, hæmoglobin value was 60%, the red cells numbered 3,000,000 and the white cells 11,600 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 29%, large lymphocytes (including some large mononuclear cells) 27%, small lymphocytes 38%, myeloblasts 6%.

Anisocytosis was extreme, with numerous macrocytes and nucleated red cells, some megaloblastic, but chiefly normoblastic, were more numerous than in any previous film.

On January 5, 1921, the hæmoglobin value was 72%, the red cells numbered 3,650,000 and the white cells 5,600 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 47%, eosinophiles 5%, large lymphocytes (and mononuclear cells) 22%, small lymphocytes 18% and myeloblasts 8%. Much less evidence of erythroblastic activity was apparent in the blood film.

On January 10, 1921, the red count was at its highest

level, totalling 4,800,000 cells in each cubic millimetre, with the hæmoglobin value at 80%.

On January 12, 1921, the hæmoglobin value was 80%, the red cells numbered 4,600,000 and the white cells 42,000 in each cubic millimetre.

On January 17, 1921, the hæmoglobin value was 70%, the red cells numbered 4,100,000 and the white cells 260,000 in each cubic millimetre. The differential leucocyte count showed the neutrophile poly-morpho-nuclear cells to be 3%, eosinophiles 1%, lymphocytes and mononuclear group 9%, myeloblasts 86% and myelocytes 1%.

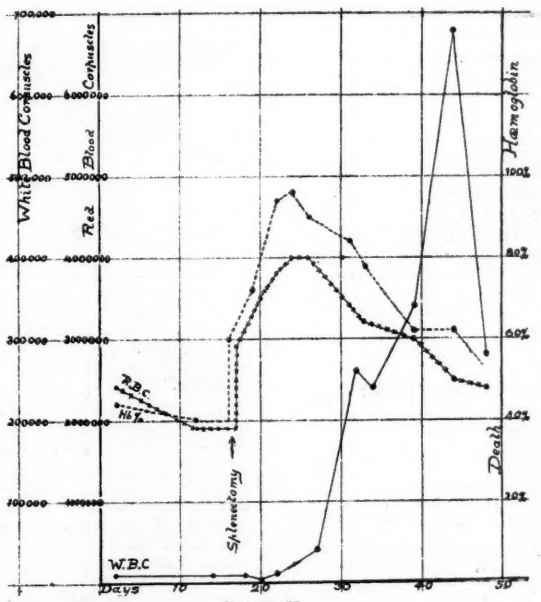
On January 19, 1921, search for enlarged glands revealed the presence of small, shotty glands in groins and axillæ and rather larger and softer glands in sub-maxillary and sub-mental regions.

During this period the child had steadily recovered from the operation and by this time was walking about the ward, enjoying a full diet and she looked very well.

On January 24, 1921, X-ray treatment was instituted. She was to have a full pastille dose of hard rays, filtered through a quarter of a millimetre of aluminium, twice a week in rotation to the following areas: abdomen, right axilla, left axilla, right groin and left groin.

By January 28, 1921, her clinical condition corresponded with the blood picture; the glands in the neck and axillæ were increasing in size and those in the groins were easily palpable. Small bruises were present about her knees and her stools were streaked with bright red blood in small quantity. Vomiting and acetonuria, bleeding from the gums, hyperpyrexia and rapid pulse rate, with progressive anæmia, leuchæmia, asthenia, glandular enlargement and nodular subcutaneous hæmorrhages culminated in death on February 5, 1921.

The progressive red blood corpuscle and leucocyte counts and estimations of hæmoglobin are presented in Graph II..



GRAPH II..

This graph presents the white blood cell and the red blood cell counts and the hæmoglobin estimations. At the point indicated by an arrow splenectomy was performed and a transfusion of blood was made. The patient died two days after the last recorded blood examination.

Autopsy.

Subcutaneous hæmorrhages with small, hard centres were most numerous about the lower limbs and were regarded as emboli. Multiple infarction, especially in, but not confined to, the posterior portions of the lower lobes had occurred in the lungs. The heart muscle was pale and rather friable, but the valves were of normal appearance. The liver was much enlarged and showed advanced fatty spoiling. The kidneys were large and very pale, with some loss of markings. The left kidney contained one infarct about one centimetre square. The stump of the spleen pedicle was soundly healed. The tracheo-bronchial and hilus glands were enlarged and hæmorrhagic. The mesenteric and retro-peritoneal groups of glands were greatly enlarged and pale, though some glands of the latter group were typically "hæmo-lymph" glands. The bone marrow in the centre of the shaft of the femur was dark red and very soft. A smear showed a great preponderance of the myeloblastic type of cell.

A mesenteric gland which was examined microscopically was found permeated with myelogenous cells and the whole structure was more loosely knit than a normal lymphatic gland and no germ centres had survived. The myeloid tissue in this and in other glands examined included erythroblasts, very large megakaryocytes, myelocytic and poly-morpho-nuclear forms.

Points of Interest.

1. The completeness of the notes, especially those covering the period of some four months before the nature of the disease was evident.
2. The insidious onset and the complicating sepsis and infective diseases.
3. Evidences of hæmatopoietic instability and the utility of blood transfusion.
4. The diagnostic importance of a low percentage of neutrophile poly-morpho-nuclear cells, even in a small total count, and the presence of myeloblasts reported as early as September 15, 1920.
5. The terminal great rise in the white count preceded clinical manifestations by at least a fortnight.

Acknowledgements.

Dr. Reginald Webster and Dr. S. O. Cowen, the Hospital Pathologists, carried out practically the whole of the laboratory work in connexion with this report.

Reviews.

X-RAY DOSAGE.

A HANDBOOK by Drs. Witherbee and Remer on X-ray dosage[1] is a collection of the valuable papers published by the authors in the various journals of America during the past four years.

The methods described are safe for spark gap readings of from seventy-six to two hundred and fifty-four millimetres and for distances between fifteen and sixty-six centimetres. The authors have had no experience of "high voltage" work. The authors deal, firstly, with unfiltered dosage; secondly, with filtered dosage; and, thirdly, with the various conditions which are benefited by radiation, while a valuable chapter is included on the prevention of X-ray burns in radiography.

The methods of measurement are based on the actual experimentally-produced erythema dose and reference to pastilles is only made for methods of comparison.

Four fundamental factors determine dosage, *viz.*, voltage or spark gap, current or milliampèrage, time and target-skin distance. With transformer apparatus, all these factors can be accurately measured.

By actual measurements the authors have found that with a spark gap of seventy-six millimetres, a milliampèrage of three and a twenty centimetre skin distance with four minutes' exposure, an erythema dose is produced with only temporary alopecia; this corresponds to the usual Sabaraud *B* dose and this combination of factors can be expressed as a fraction and any other combination of factors can be expressed as a fraction and its relation to the erythema dose determined. Doubling any one factor

[1] "X-Ray Dosage in Treatment and Radiotherapy," by William D. Witherbee, M.D., and John Remer, M.D.; 1922. New York: The Macmillan Company; Sydney: Angus & Robertson, Limited; Crown 8vo., pp. 87. Price: 8s. 6d..

doubles the dosage, whilst halving any factor halves the dosage. By this method the safety of projected radiographic exposures can be determined and the authors give a list of "safety" exposures of various regions; the insertion of 0.5 millimetre of aluminium allows of three times the number of plates being taken of any region.

Filtered dosage varies from unfiltered dosage and dosage formulæ have been prepared for the various filtrations, as it is found that filtered rays do not obey the "law of inverse squares," the dosage at the half distance being only twice as great as that at full distance, instead of four times as great.

The authors report great success in the treatment of enlarged tonsils and adenoid vegetations and claim that by this method of treatment operation for removal of these organs is seldom necessary. Eight applications are given at intervals of two weeks with five milliampères, twenty-five centimetres distance, 17.8 centimetres gap and four minutes' exposure with a three millimetre aluminium filter.

The authors also advise radiation of the tonsil areas in goitre therapy.

This book is a most valuable addition to X-ray literature and should prove invaluable to those engaged in the lower voltage therapeutic radiations.

MEDIÆVAL MEDICINE.

. . . . "A DOCTOR OF PHYSIC.

In all this world ne was there none him like
To speak of physic and of surgery.
. . . . . .
He knew the cause of every malady,
Were it of cold or hot or moist or dry
And where engendered and of what humour.
He was a very perfect practiser,
The cause yknow and of his harm the root
Anon he gave to the sick man the boot.[1]
. . . . . .
His study was but little on the Bible,
In sanguine and in perse[2] he clad was, all
Linéd with taffat' and with sendall[3]
And yet he was but easy of dispence.
He kept that he won in the pestilence,
For gold in physic is a cordial,
Therefore he lovéd gold in special."

Such words from Chaucer, the father of English poetry, might well have been written of John of Arderne, the father of English surgery. Both were attached to Old John of Gaunt, "time honoured Lancaster," from whose loins were derived royal families in England, Scotland, Castile, Portugal, Sweden.

During his life, 1307 to 1390 (?) were troublous times. The glorious age of chivalry still survived under Edward III. and the glamour of the crusades were still in men's minds. During the Hundred Years' War, which drained England of much of her best blood, the British Empire, which under Henry II. had extended from Aberdeen to the Pyrenées, dwindled in France to a single foothold at Calais. In 1349 the plague visited England. This greatest pestilence of the western world since Roman times decimated the population. The cemetery near St. Bartholomew's had a few years later to be extended; fifty thousand plague bodies have been buried there. The pestilence tolled the death-knell of feudalism and heralded grave social disturbances. Wycliffe, lecturer at Oxford, planted the seeds of the Reformation in England. The common people rose in futile rebellion to the cry: "When Adam delved and Eve span, who was then the gentleman?" The first *Medical Act of 1363* compelled every surgeon to belong to a guild—the mediæval "B.M.A.."

The Hundred Years' War gave a very high status to the military surgeons, of whom Master John Arderne was the *doyen*. Previously the prohibition by the church against clerics shedding blood had tended to divorce surgery from medicine and hand minor operations over to the barber.

[1] Remedy. [2] Red and blue. [3] Thin silk.

The guild of (military) surgeons (*magistri chirurgorum*), as clerics of the long robe, were for a time responsible for the training of the lay barber surgeons (*magistri barbi tonsores chirurgiam exercentes*), who were more or less male nurses, cupping, bleeding, blistering, giving enemata, extracting teeth, etc..

As a physician John Arderne followed the school of Salerno, with a bias towards astrology, charms and folk medicine and herbal lore.

In surgery he was up-to-date, aseptic, using alcohol and avoiding irritants to wounds, enforcing cleanliness of sutures and needles, the surgeon's nails and hands and instruments. Both in diagnosis and treatment of rectal conditions he was remarkably modern. The present operative method for *fistula in ano* was invented and practised by him. In training, experience, status, personality and good character he was worthy to be the pioneer of English surgeons.

A copy, with translation by D'Arcy Power, of an old manuscript in the Stockholm Museum—a posthumous copy—of his "*De Arte Phisicali et de Cirurgia*" has now been published by the Wellcome Historical Medical Museum, London, as No. 1 in their "Research Studies in Medical History."[1]

Of distinctly amusing interest are the clever, but quaint, thumb-nail marginal sketches by an unknown artist, drawn especially from the surgeon's rectal cases. The curious, frog-like diagrams of the human body represent the traditional convention. The scientific study of anatomy was in the embryonic stage, waiting to be exhibited in full, modern guise under Vesalius, born two centuries later. The fairy-like manikins dancing lightly in transparent balloons demonstrate the fanciful ideas on the reproductive processes.

The document itself is a systematic treatise, dealing with various ailments, beginning with the hair of the head and ending with poisonous bites and *fistula in ano*. He relates a case of the latter cured by free incision in twelve weeks. The patient was apparently satisfied "and he said, indeed, that twenty doctors had treated him and he had been operated on by ten. Nevertheless he rode on horseback about the fortieth day after I made the incision."

Arderne's fee for operation on fistula was not less than £100 in present-day money and ran up to £500 and robes and fees of £100 per year from a "worthyman and a great."

On the medical side faith played or had to play an important part. "Against epilepsy write these three names with blood taken from the auricular finger (little finger) of the patient—Jasper, Melchior, Balthazar (*i.e.*, the three wise kings from the east) and put gold, frankincense and myrrh into a box. Let the patient say three *pater noster* and three *ave maria* daily for the souls of the fathers and mothers of these three kings for a month and let the patient drink for a month of the juice of a peony with beer or wine . . . and without doubt this remedy never fails." Modern approval would probably be confined to the American States.

A tooth which it was desired should be removed: "Rub the tooth with the gum of ivy and it will immediately fall out or even if it be applied to the tooth, but be careful not to touch the other teeth."

Despite these curious bygone astrological or herbal fancies, Arderne's fame depends on his surgery. His manuscripts were widely read in England for three centuries at least, thanks to their acute observation, high moral tone and essential practical common sense.

The first publication of the Historical Museum is admirable. Let us hope that more of the series will soon appear and make available to overseas members these most interesting and fascinating medical and surgical historical originals.

[1] "De Arte Phisicali et de Cirurgia of Master John Arderne, Surgeon of Newark, Dated 1412." Translated by Sir D'Arcy Power, K.B.E., M.B. (Oxon.), F.R.C.S., from a transcript made by Eric Millar, M.A. (Oxon.), from the Replica of the Stockholm Manuscript in the Wellcome Historical Medical Museum; 1922. London: John Bale, Sons & Danielsson, Limited; Crown 4to., pp. 60, with coloured frontispiece and thirteen plates. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 18, 1922.

The Control of Tuberculosis.

IN the year 1921, according to the Commonwealth Statistician, 3,105 persons died of pulmonary tuberculosis in Australia. In comparison with the countries of the old world this figure might be regarded as satisfactory. In the course of the past five years the number of deaths from consumption has not decreased. In 1917 it was 2,882; in 1918 it was 3.038; in 1919 it was 3,392 and in 1920 it was 3,078. It is therefore not a satisfactory position, for modern doctrines would suggest that the mortality of a preventible disease should decrease steadily and rapidly. The mortality of pulmonary tuberculosis last year was 57.48 per hundred thousand of the population.

Hygienists and the public health authorities, when discussing the tuberculosis problem, usually congratulate themselves or the community on the great reduction in the tuberculosis death-rate over a long period of years. It is probable that the activities of the public health authorities during what may be termed the sanitary reform period contributed to a large extent to this improvement. Of the other factors, the industrial revolution, as it has been termed, cannot be ignored. The elimination of low wages, the improvement of the conditions of the worker, the shortening of the hours of labour and the general enhancement of prosperity are elements of a high hygienic value. It would seem that the steady decline in the incidence and mortality of tuberculosis, not only in Australia, but in all countries, was determined by the improved housing conditions, the diminution of abject poverty and dire want, the reduction in alcoholism and the greater attention paid to the health of the individual. Then came the war. And in its train came the herding together of immense masses of men, the constrained existence of predisposed individuals in an environment of wetness, cold and dirt, with insufficient or improper nutriment, the alternation of exhausting effort and enforced idleness, the strain and excitement of personal risk and the continuous reminders of injury and death, together with many other personal factors of moment. The immediate result was a sudden increase in the number of deaths from tuberculous disease. In Great Britain the years 1915, 1916, 1917 and 1918 were signalized by a considerable rise in the death rate from pulmonary tuberculosis. But since the soldier victims were young men whose war service determined the lethal outcome of an existing infection or provided the exciting cause in the presence of a predisposition, the harvest of deaths materially reduced the number of the predisposed and thus led in the post-war period to a striking reduction below the pre-war level of the mortality rate. The lowest point was reached in 1920. It is a significant fact that the mortality among females during the war years also underwent an increase, although to smaller extent than among males. It must, however, be remembered that many women took part in the campaign in Flanders, France, Egypt, Gallipoli and the other theatres of war, that an immense army of female industrial workers arose and that these women were exposed to unaccustomed influences which ordinarily have an ætiological import in connexion with tuberculosis in the male, that the food restrictions exerted a baneful effect on "the weeping maids at home," the combination of insufficient food and mental anguish, the waiting for news, lowering the resistance to the infective process. In London the death rate from pulmonary tuberculosis rose from 130 per hundred thousand of population in 1913 to 170 in 1917 and reached 106 in 1920. That starvation, mass poverty, the lack of reasonable home comforts, the absence of medical and social necessities can create a wholesale spread of tuberculous infection is demonstrated by the figures from central Europe. In Warsaw the mortality was 300 per hundred thousand of population before the war. In 1917-1918 it reached the appalling figure of 840, but in 1920 it was still above the pre-war level. In Vienna the peak was not reached until 1919.

In Sir George Newman's annual report as Chief Medical Officer of the Ministry of Health, mention is made of the fact that in the decade 1851 to 1860

the death rate from all forms of tuberculosis was 348 per hundred thousand of population, while in 1913 it was 135. For the sake of comparison it may be well to mention that the mortality from pulmonary tuberculosis in 1921 was 69.3 per hundred thousand of population. The Australian figure of 57.48 loses some of its glamour. The better social conditions of the worker, the health-giving sunshine, the absence of overcrowding, the smaller incidence of the effect of war and its concomitants on the general population and greater amount of indulgence are sufficiently important to justify the conclusion that the mortality in Australia should be lower than it is.

The problem can be approached in another way. Pulmonary tuberculosis is a notifiable disease in every State except New South Wales, where it is notifiable only in the metropolitan area, the Hunter River district, the Blue Mountain Shire and the Katoomba district. During 1921 4,214 notifications of pulmonary tuberculosis infections were received by the health authorities of the States. No one will accept this figure as the true index of the incidence of the disease. It is usually held that for every person dying of tuberculosis there are between four and five alive with signs of the disease. In the course of a very important experiment in Framingham, in the American State of Massachusetts, to which reference will be made later, it was discovered that the ratio of infected persons living to those dead of tuberculosis was 9:1. In 1921 there were 3,105 deaths from pulmonary tuberculosis registered in the Commonwealth. No doubt there were many more, since some people wish to suppress the fact that a relative has died of this disease and under the conditions of death registration this can be done at times. In addition a medical certificate of death is not given in every instance and errors of diagnosis are probably common. But even if the figure be accepted, it must be assumed that there are at any given moment in Australia between five and ten times that number of persons alive with the disease. In any case the notification figures are of small value for statistical purposes. But the object of notification is to enable the responsible health authority to locate infection and to take steps to limit or remove the danger of the sources of infection. In the case of tuberculosis the infective agent is probably less important than the other ætiological agents. Sir George Newman calls attention to the fact that in various towns and cities a large proportion of the infections from patients who had died, had not been notified at all. For example, at Barnsley the infections of 85% of the persons who died in 1921 of pulmonary tuberculosis, had not been notified or had been notified shortly before death. He regards this neglect or delay on the part of the medical practitioners as a serious matter, inasmuch as it prevents the adoption of those measures that are held to be necessary to combat the disease. In this connexion reference should be made to the fact that the tuberculosis scheme has recently undergone a change in Great Britain. Prior to May 1, 1921, sanatorium benefit was a function of the National Insurance Commissioners and the onus of making provision was borne by this organization. Since that date each County Council or County Borough Council has been required to provide a scheme for the treatment of tuberculosis within its area. It would seem that under the new arrangement, as under the sanatorium benefit, the chief reliance is placed on the institutional treatment of patients in the early stages of the disease, the isolation of persons with advanced lesions and the well-planned after-care, with suitable occupation of the improved. General prophylactic measures are carried out by health visitors. Sir George Newman finds that effective control necessitates a close co-operation between the tuberculosis officers and the general practitioners. In England there were last year 339 tuberculosis officers and 443 sanatoria and other institutions with an aggregate number of 18,943 beds. The cost of maintaining these schemes was £1,644,017, exclusive of treatment provided for ex-service men by the Ministry of Pensions. It is difficult to ascertain how much money is spent in Australia in the endeavour to check this world-wide scourge.

A few years ago the Metropolitan Life Insurance Company of New York recognized that the cost of tuberculosis was much too high. In the year 1915 no less than four million dollars were paid to 14,325 persons insured under their industrial scheme. The company therefore determined to institute an experi-

ment. The American Association for the Study and Prevention of Tuberculosis was approached with the request that an experiment on a large scale be undertaken. The sum of $100,000 was given for the purpose for a three years' period. Later a like sum was added, in order that the experiment might be extended for a further period of five years. The American Association for the Study and Prevention of Tuberculosis selected the town of Framingham, which has 16,000 inhabitants. A staff of fourteen members, including several practitioners well versed in tuberculosis work, started the campaign in September, 1916. The control was placed in the hands of a medical director and organizer, while the diagnosis work was entrusted to a skilled expert. There was no compulsory notification of tuberculosis. It was therefore necessary to institute a plan of voluntary submission to examination. The organizer used commercial methods of advertisement to inform the people that free medical examination was obtainable and invited every citizen to take advantage of the offer. A large number of medical practitioners were requisitioned for the systematic examination of the people, aided by a large staff of visiting nurses. During the first year no less than 4,473 individuals submitted to examination. No treatment was undertaken. When a person was found to be in need of treatment, he was referred to his usual medical attendant or to the public clinic. Only 23% of the people examined were found to be in perfect health. Much important information was revealed by this examination. For the present purpose it is sufficient to note that tuberculosis was discovered in 2.16% of the examined population. Later a much larger number of persons was examined. In November, 1918, three hundred and eleven persons infected with tuberculosis were discovered. The disease was in an active stage in sixty-six and in a latent stage in one hundred and fifteen. Those in whom the diagnosis was not definite, were kept under observation, while the others were under treatment in their homes or in hospital. The daily newspapers lent valuable aid by the publication of leading articles each week and in other ways. The people were asked to consider why it was that machinery is subjected to regular inspection and even motor-cars are overhauled at regular periods, but the delicate human machine is allowed to run without an annual examination.

In the first year nearly half the infected persons were found to be in an advanced stage of disease. As a result of the measures adopted, the proportion of advanced disease was reduced by 1921 to 17% of the infected. This means an enormous gain in early diagnosis. The mortality in the years prior to the commencement of the experiment was 121.5 per hundred thousand of population. In 1921 it was 40. This means a reduction in five years of 67%. By means of a free collaboration between the practising profession and the organizers of the experiment the tuberculosis mortality was reduced within a short period to the lowest figure yet recorded in any country. No new methods of treatment were introduced to attain this result. The infantile mortality fell in the same time by 22%. The von Pirquet test had been applied to no less than two thousand, five hundred children and those who had reacted, had been sent to holiday colonies or open-air schools. The tuberculosis case mortality during the first four years was 21%. It is too early yet to ascertain the full effect of this experiment on the incidence of the disease in Framingham, but it is quite evident that it has already been reduced to a considerable extent. It has often been claimed that tuberculosis is a preventible disease. The evidence is available. It is necessary to remember that the instigators of this experiment were the managers of an insurance company. They found the cost of tuberculosis too great. By investing two hundred thousand dollars they have demonstrated that the economic loss caused by tuberculosis can be reduced. The Metropolitan Life Insurance Company of New York will be in a position in 1924 to determine whether it is a sound business proposition to continue an annual expenditure of money to lessen the burdens of compensation for incapacity resulting from this disease. The Framingham experiment is merely a demonstration. But it has a far-reaching significance. On another occasion this significance will be examined in these columns, with the object of ascertaining whether a practical proposal can be evolved for the material reduction of the incidence and mortality of tuberculosis in the Commonwealth.

POST-OPERATIVE MASSIVE COLLAPSE OF THE LUNGS.

MASSIVE collapse of the lung has for many years been recognized as one of the complications which may follow the performance of surgical operations. In the vast majority of instances this pulmonary condition has followed operations performed on the abdominal cavity. Rose Bradford, however, showed that pulmonary collapse might be secondary to operations and injuries of other regions. He reported its incidence after gun-shot wounds of the thigh, buttocks and ilium.

In 1908 W. Pasteur focussed attention on massive collapse of the lungs. Up to that time it had been neglected from the clinical standpoint and nothing accurate had been determined of its nature and mechanism. Pasteur had studied the condition for many years, having first described collapse following on post-diphtheritic paralysis as early as 1900. He drew attention to the findings of Keith and Halls Dally in regard to the musculature and movements of the thorax in respiration. These observers laid stress on the interaction and interdependence of the functional activities of the diaphragm and the intercostal muscles and of the movement which takes place at the manubrio-sternal joint.

Pasteur recognized two main forces in respiratory movements, the diaphragmatic and the costal. He pointed out that when the costal force was thrown out of action, apical signs of collapse were liable to appear and that when the diaphragmatic force was *hors de combat*, collapse, if present, would be manifested in the basal areas. He held that collapse could only take place when the normal muscular force which keeps the lungs in a state of expansion, was reduced to such an extent that the elasticity of the lungs was allowed to take charge. For this reason it was common to meet with collapse in patients suffering from profound paralysis and especially paralysis of rapid onset.

Pasteur distinguished clearly between massive collapse due to muscular paralysis and lobular or patchy collapse due to obstruction of the air passages. He pointed out that the latter form of collapse might involve a whole lobe and quoted a case reported by Samuel West to prove this statement. He stated that massive collapse was essentially an active process in that the elasticity of the lung was the active agent brought into play. Lobular or patchy collapse, on the other hand, he regarded as a passive process in that the retained alveolar air behind the bronchial or bronchiolar plug was passively removed by absorption.

In his investigations of the ætiology of the condition Pasteur made careful endeavour to discover causes associated particularly with abdominal operations. He drew attention to the suggestion that in some instances prolonged pressure on the diaphragm might cause interference with its action. He also referred to the suggestion put forward by Moynihan that rectal injection of large quantities of saline solution in the treatment of shock might act as a predisposing cause. He finally concluded, however, that the determining cause of massive collapse was a reflex inhibition of the muscular movements associated with the respiratory act.

Elliott and Dingley in 1914 reported eleven cases of massive pulmonary collapse occurring in one surgical clinic. All the patients had been subjected to abdominal operations. They drew attention to the fact that in all their patients there was an accompanying muco-purulent expectoration. They regarded the lesion occasioning this secretion as the causative factor. In other words, they admitted the reflex action, but thought that its source lay in the lungs or pleura above and not in the organs below the diaphragm. They drew attention to the fact that abdominal lesions before operation often occasioned much rigidity and abdominal disturbance, but were not productive of pulmonary collapse. In this way they rejected Pasteur's explanation of the ætiology.

Rose Bradford in 1919 investigated the question of massive collapse of the lung after studying its incidence following gun-shot wounds, particularly of the chest. He pointed out that if obstruction to a bronchus was the primary cause (he stated that no other pulmonary condition could be a causative factor), it was difficult to explain how a unilateral injury limited to the chest wall in a healthy man could within twenty-four hours produce an obstruction to the main bronchus of the opposite side sufficient to cause collapse of the lung, with no urgent symptoms and with no accompanying expectoration.

He pointed out that collapse could occur without any bronchial obstruction being discoverable at *post mortem* examination. He showed that there was no evidence that blocking the bronchioles could produce massive collapse. In smaller areas of collapse associated with bronchitis a certain amount of compensating emphysema is usually present and he drew attention to the fact that emphysema is not found in massive collapse. In discussing Pasteur's theory Rose Bradford stated that collapse was known to occur in a patient in whom a constrained posture or prolonged recumbency had been necessary. He held that Pasteur's theory could be accepted if the chest became fixed in an extreme expiratory phase, for in such a case the air present would be absorbed by the blood stream. In this way he combined Pasteur's conception of active and passive collapse.

Dr. F. J. Hirschboeck has recently reported three cases of massive collapse of the lung.[1] All three patients had been operated on for abdominal conditions. Two recovered and one died. No mucous expectoration was present in any of the patients. All had fever and in the two who recovered, moist râles were audible over the affected areas while recovery was taking place. In the patient who died, the bases of both lungs were involved and the affected areas were shown experimentally and microscopi-

[1] *The American Journal of the Medical Sciences*, August, 1922.

cally to contain no air, blood or pus in the alveolar spaces.

In discussing the ætiology, Dr. Hirschboeck expresses the opinion that, although various factors may produce the condition, they never act singly. His conclusions in this regard are very similar to those of Rose Bradford and when due consideration is paid to the histories, clinical findings and *post mortem* appearances met with in the instances reported in the literature on the subject, it must be held that such conclusions are probably correct.

BACTERIOPHAGIA.

THE present-day conception of infective disease is based on a twofold doctrine of a specific causal micro-organism, a bacterium, a protozoon or a protist, and a complex immunity mechanism on the part of the host. The problem of the relation of bacteria to disease presents many difficulties. The question of the reaction of the infected organism to the infecting micro-organism is also replete with anomalies and unsolved puzzles. It will be remembered that many years ago Ehrlich found it necessary to postulate the existence of a half a dozen and more hypothetical immune substances in order to explain one phenomenon, although at first the reaction seemed to be relatively simple. Recently a phenomenon has been discovered and described by d'Hérelle which appears to introduce a fresh complication into the doctrines of immunity. It was found that if an emulsion of the fæces of a person convalescing from dysentery was filtered through a close porcelain filter and the filtrate added to the turbid suspension of dysentery bacilli, the suspension became clear and the bacilli were killed. The mixture could be filtered and the filtrate again added to a fresh suspension of bacilli with the same result. This process could be repeated innumerable times.

The intensity of the reaction increased at first until a maximum was attained, but apparently the process could be repeated hundreds of times without exhausting the effect. The reaction was found to be specific, so that the filtrate from the fæces of a person recovering from bacillary dysentery of the Flexner type will act with Flexner bacilli, but not with other forms of bacteria.

The explanations offered have been varied. In the first place d'Hérelle came to the conclusion that the stools of convalescents contained a filter-passing organism endowed with the power of destroying dysentery bacilli. Others regarded the phenomenon as a manifestation of a special ferment which associates itself with an autolytic ferment derived from the bacilli and in this association possesses the power of destroying the bacilli. In the next place it has been suggested that the dysentery bacilli undergo a modification under the influence of the leucocytes and possibly other body cells, leading to the production of a substance that acquires the power of dissolving the bodies of the dysentery bacilli.

According to Dr. Carl Prausnitz[1] the ferment or dissociation product hypotheses are untenable. He has carried out a series of experiments which he quotes in favour of the assumption of an ultra-microscopical virus. In judging his work it is essential to resist the suggestion contained in his method of production. He indulges in the well-worn expedient of arguing in terms of the hypothesis he desires to prove.

If a small quantity of the filtrate of fæces be mixed with bacilli and the mixture be spread on the surface of an agar plate, clear fields without bacterial colonies appear. These clear fields can be reduced in size and number by adjusting the proportion of filtrate to bacilli. When they assume a rounded form, he refers to them as bacteriophage "germs" or colonies.

The filtrate of the fæces possesses a certain amount of colloidal dispersion. The filtrate loses little of its power by passage through an ordinary bacterium-proof Berkefeld candle. Experiments were performed to ascertain the approximate size of the particles of the filtrate. It was found that blocked filters that resisted the passage of collargol solution, held back the particles of the filtrate, so that the size of the particles would be of the nature of 20 $\mu\mu$. This is considerably greater than the size of gelatine particles in solution. In the next place Dr. Prausnitz found that mild disinfectants of the naphthalin group, especially tetralin, destroyed the action of the filtrates, but did not affect ferment action.

He then prepared an anti-bacteriophage serum in rabbits and found that this serum possessed the power to agglutinate dysentery bacilli and to fix complement in the presence of these bacilli. By treating this serum with dysentery bacilli of the same strain, the agglutinins and complement-fixing antibodies were absorbed, while the anti-bacteriophagic properties were undisturbed. Further experiments with this serum led to the discovery that when the serum and the filtrate are mixed in such proportions that the activity of the filtrate is nearly but not quite inhibited, the "subculturing" of the clear fields from plate to plate yielded a "strain" that was serum-fast.

Dr. Prausnitz describes his experiment in terms of his interpretation, instead of in terms of observed facts. He does not mention whether the rounded clear fields of agar were emulsified and filtered with each transference to a new agar plate. If precautions were taken to prevent the carrying over of dysentery bacilli, his interpretation would be highly plausible.

From the available data it may be concluded that the d'Hérelle's phenomenon of bacteriophagia probably depends on the presence of a filter-passing virus which is inimical to the bacterium responsible for the infection. It requires but little imagination to discern many interesting possibilities in connexion with the process, if this interpretation be true.

[1] *Berliner Klinik*, August 12, 1922.

Abstracts from Current Medical Literature.

MEDICINE.

Chloride Retention.

S. L. DE JONG AND A. JACQUELIN (*La Presse Médicale*, July 15, 1922) relate their experiences of the effect produced by administering sodium chloride to patients with pulmonary fibrosis or chronic bronchitis and latent or evident cardio-renal disease. Firstly, a small dose of sodium chloride (1.5 grammes daily) was given and then this was increased to ten grammes *per diem* for four days. These doses were sometimes alternated every four days. Of sixteen patients with cardio-renal disease and chronic broncho-pulmonary lesions ten showed an exaggeration of their pulmonary symptoms, dyspnœa, increase of râles and expectoration. Of nineteen patients with chronic bronchitis, pulmonary fibrosis or asthma without evidence of cardio-renal changes, four showed œdema and respiratory distress after the administration of sodium chloride and nine showed increased dyspnœa or alteration in the quality or quantity of sputum; paroxysmal dyspnœa occurred in six patients. These facts tend to show that moderate doses of sodium chloride aggravate the respiratory symptoms of chronic bronchitis, pulmonary fibrosis and asthma, especially when complicated by cardio-renal disease, and they emphasize the necessity of excluding sodium chloride from the diet of patients suffering with these diseases.

Lead Poisoning.

M. BARRON AND H. C. HABIEN (*American Journal of the Medical Sciences*, December, 1921) have investigated the histories of four patients who died from lead encephalopathy due to the use of face powder and have reviewed the literature on this subject. The four patients mentioned were members of one family, the symptoms being headache, vomiting, colicky pains, constipation, convulsions and coma, followed by death. The blood showed marked anæmia and very numerous basophile granules in the red cells. *Post mortem* examination of one patient showed the presence of lead in the liver and kidney and no other marked pathological change except wasting and a blue line on the gums. It was found that all the patients had made use of a face powder known as "Flake White" for more than a year. On analysis, this powder was found to be finely ground lead carbonate. The authors also report one non-fatal instance of lead poisoning and they refer to two other patients suffering from plumbism due to the same powder whose histories were published by T. W. Holland forty years ago. The frequency with which the diagnosis of lead poisoning is missed, is alluded to. Notes on the histories of six patients suffering from chronic lead poisoning admitted to the University Hospital, Minneapolis, are given. In four of these instances a wrong diagnosis was made before admission. The importance of cosmetics as a cause of lead poisoning is strongly emphasized.

Acute Cerebellar Ataxia.

J. P. C. GRIFFITH (*American Journal of the Medical Sciences*, December, 1921) reviews the literature on acute cerebellar ataxia. The article deals with thirty-one examples of the condition, which are all he has been able to find in journals up to the present time. The onset was acute in every instance and occurred at an early age (a few months to ten years). The attack nearly always followed an acute infection, particularly measles. The first symptoms were generally unconsciousness and absence of speech lasting a few days. Subsequently the mentality appeared to be not quite normal; there was imperfect enunciation, with ataxia of the limbs, head or trunk, staggering gait or inability to walk. Convalescence was slow and recovery eventually fairly complete, except for some psychic symptoms. Nystagmus occurred in the case of ten patients, strabismus in five. The reflexes were usually normal. Only two autopsies were obtained. There was atrophy of the cerebellum in each instance. The possible relationship of this condition to epidemic encephalitis is interesting. The author considers it to be an example of cerebellar encephalitis.

Bronchial Asthma.

W. STORM VAN LEEUWEN AND H. VAREKAMP (*Lancet*, December 31, 1921) have tried the effect of the treatment of patients suffering from bronchial asthma and hay fever by subcutaneous injections of tuberculin. They have published their results in thirty-eight cases. In thirty patients suffering from asthma and two suffering from hay fever, skin tests made by the von Pirquet method showed definite reactions. These patients were given injections of Koch's T.O.A., beginning with one cubic centimetre of a 1 in 100,000 dilution. They were confined to bed, given protein-free diet and inhalations of benzyl benzoate. Adrenalin injections were used for three or four days to cut short attacks. Injections were given at weekly intervals for two or three months. At first the dose of tuberculin was rapidly increased, but later small doses were used throughout. Except when the asthma was complicated by chronic bronchitis, the asthmatic attacks disappeared after the first two or three doses of tuberculin. Of these asthmatic patients, 78% were cured, 18% were improved and 4% showed no change.

The Relation of Syphilis to Cardiac Disease.

WILLIAM STOBIE (*The Quarterly Journal of Medicine*, October, 1921) has made an attempt to determine the relationship of syphilis to the ætiology of heart affections. The predominant cause of heart disease is agreed to be acute rheumatism. In one hundred cases of cardiac disease, Lewis obtained a previous history of acute rheumatism or chorea in 61% and of tonsillitis in 6%. The same author investigated sixty-six cases of aortic disease with a view to determining the part played by syphilis in the ætiology. Only eight of the sixty-six patients showed a response by the blood to the Wassermann test or gave a history of syphilis and these were for the most part men over forty years of age. Stobie found that of eighteen cases of aortic disease, in thirteen there was a history of syphilis or a response by the blood to the Wassermann test or both. Four patients gave a history of rheumatism and the remaining one a history of scarlet fever. The author, therefore, agrees with Boyd Campbell, who insists on the intimate relationship between aortic disease and syphilis. Campbell obtained a Wassermann reaction in twenty-four of twenty-five cases. The findings were entirely different in sixteen cases of mitral regurgitation investigated by the author. In no instance was there a response by the blood to the Wassermann test. A response was obtained, however, in three out of fifteen cases of mitral stenosis. None of the patients admitted a previous luetic infection. In patients suffering from undoubted syphilis the author has observed a mild degree of the Stokes-Adams syndrome, alternation of the heart, cardiac enlargement, extra-systoles, tachycardia, arrhythmia and a systolic apical murmur. Sudden death occurred in two instances. In one patient a gumma of the endocardium was found at autopsy, in the other a thickening on the septal endocardium.

Artificial Pneumothorax.

D. P. MARAIS (*The Medical Journal of South Africa*, May, 1922) considers the indications, scope and results of artificial pneumothorax in pulmonary tuberculosis. The author discusses the progress that this method of treatment has made since its inception in 1894 and reviews the now abundant literature. Artificial pneumothorax is indicated when the disease is not controlled by ordinary methods and when the disease is mainly limited to one lung. It should also be used in the presence of severe or recurrent hæmoptysis or when unilateral excavation and progressive tissue destruction have occurred. The author holds that there is a stage in practically all cases of advancing tuberculosis at which treatment by formation of a pneumothorax is indicated. Antagonism to this method in selected cases comes mainly from those who are ignorant of its effects. He reports his results in thirty-two patients suffering from tuberculosis and in six patients who were not tuberculous. The results were good in the tuberculous, but not good in the non-tuberculous patients who suffered from gangrene, abscess or bronchiectasis. The condition was chronic in those suffering from bronchiectasis and complete collapse of the lung was difficult to obtain.

NEUROLOGY.

Anthropology and Insanity.

ALES HRDLIČKA (*Journal of Nervous and Mental Diseases*, September, 1922) states that the knowledge of the forms of insanity which existed in early times is of necessity very fragmentary. Anthropology itself did not exist until well within the nineteenth century. Next taking insanity in different existing races, he indicates that the early physicians of Australia alluded to the absence of insanity among the aborigines. But they also point out adequate reasons for this; not only was there no occasion for mental stress, no alcohol and no syphilis, but, according to custom, the maniac—believed to be possessed of evil spirits—was done away with, the melancholic allowed to do away with himself and the occasional epileptic driven forth to shift for himself and generally die. Later writers show that the most common disorders among these people are manic-depressive states, amentia, dementia and *dementia præcox*. Forms of insanity manifesting themselves in abstract cerebration, such as paranoia, do not exist, nor, without syphilis, does general paresis. Practically the same applies to the African negro. Among certain yellow-brown people (American Indians and western Chinese), despite abundant syphilis, general paresis is rare and that seen is uncoloured by grandiose ideas. But, curiously, among Chinese mariners infected with syphilis outside China and among the Japanese general paresis is common. The yellow-browns, like the Australian aborigines, do not exhibit typical high-class paranoia. In India, Burma, Siam and the East Indian islands the insanities in general are not so violent, extreme or fatal as those those among white people. Here the alcoholic insanities are practically negligible, but special psychoses occur, those due to abuse, hashish and opium, a leprotic psychosis (Japan, Hawaii), one due to pellagra (Japan) and the "running amok" of the Malayan. Among white races, Russians of the Baltic Provinces and the Ukrainians show differences in the frequence of the various forms of insanity. In Ireland the insanities are reported to be more frequent than in England or Scotland. Jews are prone to *dementia præcox*, depressions and other psychoses, but suffer little from alcoholism. Finally, the North American Indian and the Southern American negro are relatively free from insanity. Many of these facts call for investigation and explanation.

The Pituitary Gland and its Syndromes.

JEAN CAMUS AND GUSTAVE ROUSSY (*Revue Neurologique*, June, 1922) brought before the Neurological Society of Paris results of a series of experiments on the pituitary gland of one hundred and forty-nine dogs and thirty-six cats. On the question whether the pituitary gland is essential to life, as believed by Paulesco, Cushing and Biedl, the authors can show many animals which have permanently survived destruction of the gland. They maintain that when death does occur in animals that have been operated on, it is due, not to loss of the gland, but to meningitis or hæmorrhage or lesion of the third ventricle. They also hold that it is injury to extra-pituitary parts which accounts for *diabetes insipidus* and glycosuria, modification of carbo-nydrate tolerance, interference with genital functions, obesity and skeletal alterations. They draw attention to the therapeutic uselessness of pituitary administration in patients suffering from acromegaly and infantilism and to the fact that, although pituitary medication may reduce polyuria, so also may injections of antipyrine, novocaine and other substances. They point out that anatomical study of the *tuber cinereum* and *hypothalamus*, *i.e.*, parts immediately above the pituitary gland, reveals certain hitherto unrecognized nuclei or groups of nerve cells which seem to be of fundamental importance. Thus, Percival Bailey and Bremer are quoted as showing that experimental damage of these nuclei alone in dogs has induced both polyuria and a cachexia akin to the adiposo-genital syndrome of men. They record the findings in *post mortem* examinations of two human instances of *diabetes insipidus*, one associated with past meningitis and the other with abscess, which show the presence of lesions of the *infundibulum* and *tuber cinereum* with intact pituitary.

Climacteric and Age in Men and Women.

MATHIAS VAERTLING (*Neurologische Centralblatt*, May 1, 1921) points out that Kurt Mendel brought forward practical observations to prove that there is a climacterium in men, a time when the active sexual life of men comes to an end just as it does in women. The writer thinks that Mendel's view can be infallibly proved by comparing the mortality statistics of males and females. From such a comparison it becomes obvious that at no other period of life than between the ages of forty-five and sixty does the death rate of men more exceed that of women. The mortality of men is highest at the age of forty-five, which is evidence that the male climacteric takes place earlier than has been supposed. Just as in badly nourished women the menopause occurs early, so may this cause induce early cessation of sexual function in men. The degree of sexual activity also plays an important part in relation to the climacteric. An inactive sexual life in women usually brings early cessation of function, but the opposite happens in men. Another proof of the male climacterium is the fact that quality of offspring deteriorates with advancing years in the father. Finally, the belief that men have a longer youth than women is a dream and not a beautiful one; it is not years which make age, but the manner in which the years are lived.

Non-Progressive Tabes.

A WESTPHAL (*Deutsche Zeitschrift für Nervenheilkunde*, December, 1920) records the remarkable case of a woman who died at the age of sixty years and who for twenty-seven years had shown absence of tendon reflexes. Although intermittently under observation all these years, she exhibited no associated signs or symptoms upon which to establish a diagnosis. It had been necessary to exclude *tabes dorsalis* on account of the absence of the Argyll-Robertson phenomenon, of sensory disorder, either subjective or objective, of ataxia, of sphincteric disturbance and on account of the failure to obtain a reaction to the Bordet-Wassermann test either with the blood or cerebro-spinal fluid. The patient was therefore looked upon as suffering from simple congenital absence of the tendon reflexes, some instances of which have been reported. The patient's general health gave way and she died after a short illness. At the autopsy the astonishing revelation was made that the spinal cord was shrunken and that the posterior columns, as seen in specimens stained by the Weigert-Pal method were in a condition characteristic of tabetic degeneration, although not profound in degree. Therefore, the case may have been one of monosymptomatic or rudimentary tabes and the author concludes that caution must be exercised in diagnosing congenital absence of the tendon reflexes.

The Treatment of Intracranial Tumour.

H. H. TOOTH (*The Medical Press and Circular*, June 14, 1922) says that the surgical treatment of tumour of the brain is in most cases unsatisfactory and from the nature of the disease must always be so. The difficulty is that the period in the life of the tumour when radical treatment would be possible, can rarely be diagnosed. This applies particularly to the common glioma, the glio-sarcoma and the sarcoma, which make up the majority of brain tumours. Results of operations at the National Hospital (London) for ten years—1902 to 1911 inclusive—show that only four kinds of growth lend themselves to radical treatment (extirpation) with hope of success, *viz.*, endothelioma, which is usually accessible, attached to *dura* and unlikely to recur; simple cyst (unfortunately many cysts are not simple, but are degenerated gliomata); extra-cerebellar fibroma, provided a method can be devised to allow of very gradual removal (sudden removal is highly dangerous, and pituitary adenoma. Then as to palliative treatment, when extirpation is impossible and there are no localizing signs, the author advises the first stage and the first stage only of Victor Horsley's craniectomy operation. This consists in the removal of bone, preferably in the right parietal region, with an area of at least seven and a half by five centimetres. He states that this operation has been performed many times at his request, with a resulting prolonged survival period.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Walter and Eliza Hall Institute of Research in Pathology and Medicine, Melbourne Hospital, on October 4, 1922, MR. G. A. SYME in the chair.

Splenomegaly.

DR. S. O. COWEN read a paper entitled "Familial and Hæmolytic Splenomegaly." This paper was published in THE MEDICAL JOURNAL OF AUSTRALIA, November 11, 1922, page 545.

DR. H. DOUGLAS STEPHENS read a paper entitled "Clinical Symptoms of Enlargement of the Spleen in Children" (see page 575).

DR. S. W. PATTERSON read a paper written by Miss Maud Cowen and himself entitled "Bile Salts in the Blood" (see page 581).

DR. KONRAD HILLER, in opening the discussion, said that, while he had followed the papers with great interest, he did not feel competent to discuss that of Dr. Stephens, as it was concerned entirely with disorders of infancy and childhood.

He cordially congratulated Dr. Cowen, whose paper he had had the privilege of reading prior to the meeting; it was obvious that Dr. Cowen had expended a very great amount of time and had taken infinite trouble in his study of the remarkable family which formed the basis of his paper. He felt that in the future Dr. Cowen's paper would be regarded as a milestone in the progress of knowledge regarding familial hæmolytic splenomegaly and it furnished a fine superstructure for the foundation laid by Dr. J. W. Springthorpe.

Dr. Hiller said that he found three points arising out of Dr. Cowen's investigation to which he wished to draw attention for discussion.

In the first place there was the strong resemblance in the clinico-pathological findings between familial hæmolytic splenomegaly and pernicious anæmia. Dr. Cowen had emphasized this point and had quoted in illustration the case of a man formerly in the medical wards of the Melbourne Hospital. This patient had exhibited the characteristic features of hæmolytic splenomegaly, which had embraced an anæmia of "pernicious" type; he had been treated as suffering from pernicious anæmia with good results up to a point. The splenic enlargement, however, had persisted and some months later, when the anæmia again took on a pernicious form, the spleen had been removed. Following splenectomy the blood picture had returned to the normal without any arsenical treatment.

The resemblance of the blood findings in hæmolytic splenomegaly and pernicious anæmia was not difficult to understand; in both an abnormal hæmolytic agent was at work and in the first-named there was the additional factor of undue fragility of the red corpuscles.

The spleen exercised normally a certain hæmolytic function and it was suggested that in hæmolytic splenomegaly this function was enhanced. Was it not possible that the prevailing conceptions respecting the source of the hæmolytic agent in pernicious anæmia were in error and that the corpuscular destruction which characterized pernicious anæmia, was due to splenic activity?

The results of splenectomy, however, did not lend support to this hypothesis and seemed to show that elaboration of a hæmolytic agent in the spleen or exaggerated splenic function in the destruction of red corpuscles was at most a small factor in the ætiology of pernicious anæmia, in which condition temporary benefit only followed removal of the spleen. Information bearing upon the results attending splenectomy for the relief of pernicious anæmia was furnished in some figures published by the Mayo Clinic. At Rochester the operation of splenectomy had been performed fifty times upon patients suffering from pernicious anæmia; the operative mortality had been 6%. Ten of the patients who had survived the operation, had lived for three and a half and five others for four and a half years.

In view of these figures, Dr. Hiller submitted that it was not a fair claim that splenectomy was a beneficial measure in the treatment of pernicious anæmia and pointed out that it was not uncommon to see a five years' course in pernicious anæmia under medical treatment. In the article to which he had referred as emanating from the Mayo Clinic, it had been concluded that splenectomy might be recommended when in view of all the circumstances the possibility of a prolongation of life appealed to the family and to the patient; occasionally it might be resorted to in order to bring about an immediate remission.

In the second place, Dr. Hiller considered hæmolytic splenomegaly as compared with splenic anæmia. He recalled the case of a female patient who had been admitted to the wards of the Melbourne Hospital on account of ascites and who had stated that she had undergone splenectomy in England seven years previously for Banti's disease. Ascites had been present at the time of the operation, but had not recurred until seven years later. The operation of omentopexy had been advised and attempted, but it had been found that it had already been performed. A state of advanced cirrhosis of the liver, however, had been noted and it had appeared that the former splenectomy had not been carried out sufficiently early to obviate the progress of the hepatic cirrhosis.

In conclusion, Dr. Hiller contributed some remarks relative to enlargement of the spleen in general and to hæmatogenesis. He had always regarded the enlargement of the spleen in various conditions, even in malaria, as something of a mystery and had never found a satisfactory explanation for this commonly observed phenomenon. Perhaps the lack of success in this particular had been connected with the very imperfect state of knowledge in respect to the function of the spleen.

It appeared that the present conceptions of hæmatogenesis, formerly considered well based, were now to be revised. The bone marrow and lymphatic glands had always been regarded as the sources of the blood cells, the former contributing red cells and leucocytes of the granular series and the latter supplying the lymphocytes. Such a view appeared quite consistent with the hyperplasia of the lymphoid tissues and the blood picture observed in lymphatic leuchæmia and while it referred the blood changes in spleno-medullary leuchæmia to perverted activity of the bone marrow, it failed to explain the enlargement of the spleen.

More recent views regarding the origin of blood corpuscles were quite different from those so long accepted. Whereas it had formerly been taught that in the embryo hæmatogenesis began in the endothelial cells of the capillaries, was carried on later in the liver, still later in the spleen and eventually at birth left the spleen for the bone marrow, it was now held that the whole reticulo-endothelial system of the body was responsible for the formation of blood cells. It was being urged that there were three types of capillaries, that the poly-morpho-nuclear leucocytes were derived from the endothelium of the venous capillaries, the erythrocytes from the arterial capillaries and the lymphocytes from lymphatic capillaries. All the capillaries were to be considered as potential hæmatogenetic centres and the bone marrow provided the most favourable conditions for the observation of hæmatogenesis.

On this basis it might be asked if the enlargement of the spleen in spleno-medullary leuchæmia was due to the activity of those capillaries which produced pre-myelocytes and in polycythæmia it might be suggested that the splenomegaly was dependent on exaggerated function of the endothelium of the arterial capillaries leading to the production of an excessive number of red corpuscles. These were suggestions only and remained to be elaborated.

Dr. Cowen had shown slides with the object of demonstrating the hyperplasia of the reticulo-endothelial cells of the spleen in acholuric jaundice or hæmolytic splenomegaly. Possibly this feature indicated the essential cause of the enlargement of the spleen in hæmolytic splenomegaly.

DR. S. W. PATTERSON said that he wished to take advantage of the occasion to direct the attention of members of the Branch to the Clinical Research Scholarship during his tenure of which Dr. Cowen had carried out the work embodied in his admirable and exhaustive paper.

Dr. Patterson reviewed the history and indicated the present status of the Clinical Research Scholarship[1] and in so doing paid a tribute to the pioneer work of Dr. S. V. Sewell and Mr. Alan Newton in the establishment of the scholarship. Its present value was £100 *per annum* and it carried the privilege of a year's residence at the Melbourne Hospital and facilities for work in the Walter and Eliza Hall Institute of Research in Pathology and Medicine. He urged practitioners to consider the claims of the clinical research fund and to take every opportunity of interesting people who might be disposed to help. He spoke as a member of the committee in whom the administration of the clinical research fund was vested and expressed regret that Dr. Sewell was unable to be present.

DR. H. FLECKER inquired of Dr. Cowen, in view of his statement that non-operative treatment was of no avail, whether he knew of any patients suffering from hæmolytic splenomegaly who had been subjected to irradiation. In certain other conditions, such as spleno-medullary leuchæmia and even malaria, considerable diminution in the size of the spleen followed intensive radiation and, while Dr. Cowen's figures were very good, showing a mortality of two in a series of forty-eight splenectomies, he wished to suggest the use of X-rays as being possibly of advantage in patients affected too mildly to justify operation on for those in whom splenectomy was contra-indicated or refused.

MR. H. B. DEVINE expressed the pleasure with which he had followed both papers and remarked that that of Dr. Cowen had done much to clarify the hitherto foggy atmosphere which had surrounded splenic disorders.

In surgical exploration of the abdomen it was remarkable to note how frequently the spleen displayed an appreciable degree of enlargement. The spleen seemed to be a home in which bacteria could hibernate after their filtration from the circulation, with consequent enlargement of that organ from the stimulation of its cells by toxic action. He had also observed occasionally some enlargement of the spleen in radiographic examinations and such enlargement might be associated with the many forms of obscure *focal* sepsis. Pain in the left hypochondrium without obvious cause was probably often due to some form of perisplenitis associated with moderately enlarged spleen. Perhaps as physicians and surgeons they had not given the spleen all the attention that it merited as a possible site of obscure pathological processes.

Mr. Devine reviewed briefly the history of a woman who had come under his observation after six months of illness; she had been extremely weak and her symptoms, as well as the blood picture, had been, in the opinion of himself and the consulting physician, Dr. Latham, diagnostic of pernicious anæmia. The spleen had been very distinctly enlarged. He had performed splenectomy. The patient had made a remarkable recovery and was still well, but he had since realized that it was open to question whether he had not been dealing with a profound secondary anæmia in which embryonal cells had appeared in the blood as the result on the heavy call on the blood regenerating mechanism and this anæmia might have been more of the type that was associated with the splenic activity of splenic anæmia or hæmolytic jaundice. The enlarged spleen had seemed to suggest this.

He wished to add that this instance had been the only one in his experience in which splenectomy had apparently been successful in pernicious anæmia; in every other patient on whom he had operated, temporary benefit had resulted, but the fatal issue of the disease had not been averted. It appeared then that there were some patients with pernicious anæmia who should be given the benefit of operation, notwithstanding the poor results of splenectomy. The reason for this was that in some instances the type of the disease might approximate that of hæmolytic jaundice and in this disease splenectomy usually effected a cure. Enlarged spleen in pernicious anæmia would therefore indicate operation.

In considering the question of splenic enlargement associated with cirrhosis of the liver, Mr. Devine said that the important point in diagnosis was to distinguish between Banti's disease and cirrhosis of the liver, with secondary enlargement of the spleen. Some benefit might accrue from surgery in the former, but it could hardly be expected in the latter condition. He had performed splenectomy in isolated instances of this comparatively late stage of Banti's disease, but the results were only moderately successful and they had not been encouraging. He recollected an example of successful splenectomy in a patient with an early splenic anæmia and in that instance the patient had been admitted to hospital on account of hæmatemesis considered to depend upon the presence of a gastric ulcer. He wished to congratulate Dr. Cowen and Dr. Stephens again on the very high standard of their papers.

MR. G. A. SYME, from the chair, expressed the indebtedness of the meeting to Dr. Cowen for his admirable paper, which embodied the results of a laborious investigation and was a very gratifying outcome of the establishment of the Clinical Research Fund. He wished to endorse Dr. Patterson's remarks commending the fund to the notice of the profession and, although it was now upon a satisfactory basis, he hoped that its present development was only a beginning and that they would see many more clinical research studies such as that undertaken by Dr. Cowen.

As one who recollected the early examples of familial splenomegaly studied by Dr. Springthorpe and Dr. Stirling, he had found the paper and discussion extremely interesting. It appeared that it was now necessary to revise the original diagnosis of splenic anæmia affecting this family and in connexion with the points of resemblance between hæmolytic splenomegaly and pernicious anæmia he remembered Dr. J. F. Wilkinson drawing attention to the fact that the anæmia of familial splenomegaly was of "pernicious" type.

As Dr. Hiller had observed, the essential cause of the enlargement of the spleen in the various so-called blood disease was very obscure. Why, for instance, should the spleen enlarge in each of the two apparently opposite conditions, polycythæmia and splenic anæmia?

The diseases of the blood and blood-forming organs offered a fruitful field for research.

DR. COWEN replied briefly to the various speakers. He agreed with Drs. Hiller and Devine that the results of splenectomy in pernicious anæmia had not been at all good. In regard to the patient whose history had been detailed by Dr. Devine, Dr. Cowen was unable to say whether the diagnosis of pernicious anæmia was correct. He had, however, seen a similar case in which there had seemed to be doubt as to the correctness of the diagnosis. The patient had recovered without operation. In splenic anæmia the improvement was not always maintained, as instanced by Cushing's account of Osler's early cases. Dr. Cowen had seen relapse with recurrence of hæmatemesis and other symptoms three years after splenectomy.

Dr. Cowen hoped that Dr. Flecker's contention that deep X-ray therapy would be of use in hæmolytic jaundice was correct. He was trying this treatment on a patient at present under observation.

[1] Information concerning the Clinical Research Scholarship is to be found in the issues of THE MEDICAL JOURNAL OF AUSTRALIA of July 8 and August 5, 1922.

PRESENTATION TO DR. W. T. HAYWARD.

A MEETING of the South Australian Branch of the British Medical Association was held in the Lister Hall, Hindmarsh Square, Adelaide, on October 27, 1922, DR. T. G. WILSON, the President, in the chair.

THE PRESIDENT, in making a presentation to Dr. W. T. Hayward, C.M.G., on behalf of the members of the South Australian Branch, said that the portrait was only a small token of the appreciation of his colleagues of the work Dr. Hayward had done and of the high esteem in which he was held.

SIR JOSEPH VERCO said that it gave him much pleasure to say a few words about Dr. Hayward. Dr. Hayward had taken his degree in 1874 and in 1878 had come out to Australia. He had settled in Adelaide in 1882 and, since then, he, Sir Joseph Verco, had been a colleague of

Dr. Hayward, firstly as an Honorary Physician to the Adelaide Hospital and later as an Honorary Consulting Physician. Dr. Hayward had been on the Board of Management of the Adelaide Hospital for twenty years and everyone knew what that meant.

They were also colleagues at the University. Dr. Hayward had been on the Council for twenty years and had been Lecturer on *Materia Medica* and Therapeutics until last year. The very highest honour had been conferred on him last year, when he had been elected Dean of the Faculty of Medicine. They had been delighted to see him in his robes of honour at Commemoration.

Before his arrival in South Australia Dr. Hayward had been a member of the British Medical Association and he was one of those who had sent the request to the parent body that a branch might be formed in Adelaide. He had been a frequent contributor to *The Australasian Medical Gazette* and one of the very first men to read a paper on the treatment of compound fractures by antiseptics. Before his arrival he had worked with Thomas and he had been the first to introduce the Thomas's splint out in Australia.

In 1885 he had been elected President of the Branch. He was only thirty-one years of age at the time. Twenty years later he had again been elected to the chair. When the Federal Committee was formed, Dr. Hayward had been elected chairman, a position he had held until his resignation in 1921.

Dr. Hayward was present at the Annual Meeting of the British Medical Association in 1914 and the members did him the honour of electing him a Vice-President. On the same occasion the University of Aberdeen had conferred on him the degree of Doctor of Law.

In 1915 Dr. Hayward had enlisted for active service abroad. On his arrival in England he had been made Officer Commanding the Australian Auxiliary Hospital, Harefield. He, Sir Joseph, felt sure there were many present that night who would say that they had received kindly and useful advice from Dr. Hayward whilst in England.

The enormous amount of work which Dr. Hayward had done, would give the members some idea of the debt which the Branch owed to him. The tact shown by him in every position he had held and the quiet persistence in difficulties had assured the success of all his undertakings.

For these reasons Dr. Hayward was honoured and esteemed and Sir Joseph Verco offered him the congratulations of the Branch and of himself on the presentation of his portrait.

The portrait was then presented to Dr. Hayward.

DR. HAYWARD, in thanking the President and members, said that it was the proudest moment of his life and that he was quite overcome by the reception he had received. He thanked the members and all those who had initiated the idea. Fifty years ago he had started to earn his living at the profession. He started his course at the age of fifteen and a quarter years at the Liverpool Medical School and Sir Robert Jones had started with him a few months later. He had passed through pre-antiseptic days and through the time when Lister was doing his great work. It gave him great pleasure to accept the portrait, which he thought was an excellent likeness. He would, however, ask of the Branch another honour and that would be to accept his portrait to be hung on the walls of the Lister Hall among the distinguished portraits of those who had gone before and of those which would be hung there in the future. He thanked everyone very much.

THE PRESIDENT, in reply, said he felt quite certain the Council would be only too honoured and pleased to accept Dr. Hayward's very kind offer.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

TAHMINDJIS, GEORGE, M.B., Ch.M., 1922 (Univ. Sydney), 151, Elizabeth Street, Sydney.

Medical Societies.

THE OPHTHALMOLOGICAL SOCIETY OF NEW SOUTH WALES.

A MEETING of the Ophthalmological Society of New South Wales was held in the Sydney Hospital on February 1, 1922, DR. R. H. JONES, the Vice-President, in the chair.

Blepharospasm.

DR. R. H. JONES presented a patient who had been shown at a previous meeting of the Society. The patient was suffering from spasm of the right side of the face and of the right eyelids. The spasm had improved and he was then able to open his lids to some extent. The vision remained very defective. The prognosis of the condition was regarded as hopeless.

Coloboma of the Iris and Chorioid.

DR. F. GUY ANTILL POCKLEY presented a patient with an atypical coloboma of the iris, associated with very atypical coloboma of the chorioid. The patient was a female, nearly seven years of age, the eighth child of an apparently healthy mother, though the only child by her second husband. There was no procurable history of any affection of the eyes beyond refractive errors in the majority of the members of the father's family. It was thought that this case was sufficiently atypical to prove of interest, owing to the site of the colobomata and the nature of the ophthalmoscopic appearances. The left eye only was affected. The coloboma of the iris was almost complete and was situated in the right upper quadrant.

The ophthalmoscopic signs were very unusual. There were several clearly defined patches with some pigmentation of the margins, crossed by very scanty ciliary and retinal vessels with a surface considerably recessed below the level of the rest of the fundus. This was evidenced by parallactic movements which were regarded to be patches of a colobomatous nature. One of these areas, the largest, was several times the area of the disc and was oval, with its long axis radially placed in a situation corresponding to the iris defect.

There was another large area situated below and to the outer side of the disc; it was of a similar nature, though more irregular in shape and more circular in general outline.

There were two small, clean punched, white areas near the macula, circular, but smaller than the width of the disc in diameter; it was also at a deeper level than the surrounding retina.

All these patches, with the possible exception of the last, presented all the ophthalmoscopic appearances of typical colobomata. The retina and chorioid surrounding these large patches did not reveal any evidence of inflammatory trouble.

In addition to these areas there were signs indicative of an old disseminated chorioiditis, particularly in the macular region, where the degenerate areas and gross disturbance of pigment presented a typical appearance of post-inflammatory trouble.

Dr. Pockley said that it might be argued that all the ophthalmoscopic abnormalities might be post-inflammatory in nature, but he found that the larger areas presented such typical pictures of colobomata that there was no room for doubt on this point. It was possible to have colobomata in unusual situations, though Fuchs had taught that colobomata of the iris was always below. Werner had maintained that coloboma of the chorioid was always below.

The accepted explanation of coloboma of the iris, originally put forward by Lang and Collins, was that it was due to adhesion of the anterior fibro-vascular sheath, preventing ingrowth of iris between cornea and lens.

Dr. Pockley thought that the condition in his patient might be partly explained in two ways. Firstly, in rare cases chorioid fissure in atypical situation during fœtal life would lead to a large coloboma of the chorioid in the situation corresponding with the iris defect, but not to the other colobomata. The hypothesis that colobomata of the chorioid were due to failure of formation of blood

vessels in the inner part of the mesoblast surrounding the secondary optic vesicle, with resultant lack of short ciliary vessels (probably as the result of intra-uterine inflammation) would explain the multiple defects, but would leave unexplained the symmetry often noted in atypical colobomata of iris and chorioid. In this patient he suggested that there had been at some time a violent chorioiditis, though it must have been at a later date than the inflammation causing the coloboma.

Dr. Pockley asked other members to express their opinion in regard to his interpretation of the ophthalmoscopic appearances and also for any explanation of the pathological cause or causes which would explain the various defects in the eye.

The child was apparently normal in every other way, with one exception, *i.e.*, she was left-handed and could not be taught to write or print normally. When she had been given a pencil and had been asked to write the letters of the alphabet, she took the pencil in her left hand and, without hesitation, printed the letters from right to left in reverse, exactly as a printed line of letters would appear in a mirror.

DR. R. H. JONES discussed the case at some length and DR. J. C. HALLIDAY concurred in Dr. Pockley's diagnosis.

Traumatic Cyst of the Iris.

DR. F. J. BLAXLAND exhibited a patient with a traumatic or transplantation cyst of the iris. The patient was a woman, aged thirty-one years, who had been seen on January 14, 1922, when acute cyclitis was present. She had had an old corneal injury accidentally self-inflicted by a gimlet. This extended for three-quarters of the length of the pupil and a transplantation cyst of the iris was seen at the inner side of the pupil. An iridectomy had been done by Dr. Maher soon after injury, when the patient was nine years of age.

Mydriasis Due to an Uncommon Drug.

DR. BLAXLAND recorded a case of mydriasis following instillation of the juice of *Brugmansia knightii* (of the natural order *Solanaceæ*), of which a specimen was shown. A small boy in December last had climbed a tree and had got some of the juice in each eye. Full mydriasis had followed. His aunt, who was a nurse from the northern rivers, had introduced some of the juice in one of her eyes to ascertain its effect. Full mydriasis had followed.

Vitreous Opacity and Retinal Hæmorrhage.

DR. E. TEMPLE SMITH showed a patient, aged sixty years, who had been struck in the right eye by what felt like a minute fragment of blue metal. He had felt a sensation like "a sting." Half an hour later he could not see his hand. His sight had been good.

There was no albumin nor sugar in the urine. There was no appearance of any wound of entry in the cornea or sclera. No foreign body was detected in the eye on X-ray examination.

A floating, web-like vitreous opacity was recognized, as well as retinal hæmorrhages, one small one at the macula. The temporal part of the disc was pallid. The veins were decidedly full. His vision was "fingers" at two metres. The tension was normal. No retinal detachment was observed. The vision was markedly contracted.

The members were in agreement that the condition of the eye was partly of old standing and had not been caused by the eye being hit by a piece of blue metal.

Defective Vision After Fracture of the Skull.

DR. A. H. RUTHERFORD presented a patient, a painter, aged forty-three years, who had complained of defective sight in the right eye since an accident on November 10, 1921. He had fallen through a skylight and had sustained a fracture of the skull with right middle meningeal hæmorrhage. He had been operated on by Dr. John Morton on January 3, 1922.

The vision of the right eye was $^{6}/_{12} + 1$ $^{6}/_{6}$, and that of the left eye $^{6}/_{6}$.

The right eye deviated 10° to the right and 5° upwards. The right pupil was smaller and less active than the left.

The right eye did not converge on accommodation. The right optic disc was slightly paler than the left. Later it had become considerably paler than the left disc, which was paler than normal.

A MEETING of the Ophthalmological Society of New South Wales was held in the Eye Department of the Sydney Hospital on May 3, 1922, DR. J. J. KELLY, the President, in the chair.

Anglo-Neurotic Œdema of the Eye.

DR. TEMPLE SMITH read the notes for DR. E. A. D'OMRAIN of a case of angio-neurotic œdema of the eye.

The patient, a medical man over fifty years of age, had complained of a more or less sudden dimming of the vision of the right eye. This had been accompanied by a feeling as if the focus had been interfered with, as was produced by mydriasis. There had been a feeling extending over the region of the brow, orbit and cheek bone, as though the eye had been lightly struck with a soft ball the size of a tennis ball. This feeling had been described as a tingling sensation rather than bruising. The upper lid had become slightly œdematous, causing some ptosis.

The conjunctiva was red and injected, the pupil markedly contracted and a little fixed, accounting thereby for the blurring of images when both eyes were open, but not to any extent when the left eye was covered.

These occurrences showed some sort of periodicity, generally coming on about 7 p.m. and, after reaching a zenith, slowly subsiding in about half an hour, so that in about an hour to an hour and a half the eye reached normal again.

The attacks were sometimes daily, sometimes at longer intervals and had only occurred quite recently. There was no history of attacks in the past, but the patient in childhood had been a frequent victim of a similar condition of the upper lip, at which times the lip had swelled to a great extent. He had been always liable to urticaria.

Dr. Temple Smith had only seen one other patient with this condition, the patient being a man of about thirty years. In his case the condition had begun towards the middle of the week, had reached its zenith about Saturday and had vanished during the week-end.

The condition was said to be commoner in women at the menstrual period.

THE ANNUAL MEETING of the Ophthalmological Society of New South Wales was held in the Eye Department of the Sydney Hospital, Sir John Young's Crescent, Sydney, on August 2, 1922, DR. J. J. KELLY, the President, in the chair.

Secretary's Report.

THE HONORARY SECRETARY read his report as follows:

There were four ordinary Council meetings concerning with the general business of the Society and two special Council meetings, concerned, firstly, with an attempt on the part of the optometrists at State registration and, secondly, with certain matters arising out of letters that had been published in a weekly newspaper.

The average attendances at quarterly meetings were six out of a membership of twenty-three. Fifteen cases were demonstrated and the histories read. Accounts of eight undemonstrated cases were read. Pathological specimens of neoplasms, X-ray plates and scotoma charts were shown.

Four new members were gained during the year, *i.e.*, DR. H. J. BRENNAND, DR. A. T. DUNLOP, DR. R. SMITH CANDLISH and DR. H. MAHER; one was lost by the death of DR. GEORGE PAUL.

Treasurer's Report.

DR. R. H. JONES reported that he had received a telephone message from Mrs. Waddy, that Dr. R. G. Waddy would be unable to be present at the meeting owing to illness and asking that an apology be tendered.

Dr. Jones pointed out that, according to the Rules, the Honorary Treasurer shall present to the annual meeting a written report of the financial state of the Society, signed by himself; also that the President and Secretary shall audit the Treasurer's accounts. As this had not been done, Dr. Jones suggested that the only way out of the difficulty was to adjourn this part of the business until the next quarterly meeting.

It was decided that this course be followed.

Election of Officers.

No other nominations having been received, the President declared the undermentioned duly elected as the officers of the Society for the ensuing year:

President: DR. E. TEMPLE SMITH.
Vice-President: DR. J. J. KELLY.
Honorary Secretary: DR. F. J. BLAXLAND.
Honorary Treasurer: DR. R. G. WADDY.
Members of Council: DR. R. H. JONES and DR. CYRIL SHEPHERD.

Presidential Address.

DR. J. J. KELLY thanked the members for putting him in office for the year and bearing with his little defects. Everything had been very harmonious, as had always been the case in the Society.

He called attention to the fact that sooner or later they would have to face the opticians, who were endeavouring to get a Bill put through for their registration. He was of opinion that if every member of the Society would make notes of his cases in which treatment had not been all that it should have been on the part of opticians, it might help.

The Council of the New South Wales Branch of the British Medical Association had been interviewed in regard to the Opticians' Bill after they had been to the Government; the Council were of the opinion and the representatives of the Ophthalmological Society concurred that it would be worse than useless going to the Government at the present time, but that it would be better to wait until a Bill was drafted and then to put forward objections. The Council was in possession of various correspondence and literature dealing with the introduction of similar Bills in New South Wales and elsewhere. The New South Wales Branch of the British Medical Association had intimated that they would not go to the Government without members of the Ophthalmological Society.

DR. KELLY vacated the chair in favour of the incoming President, DR. E. TEMPLE SMITH.

DR. E. TEMPLE SMITH thanked the members for the honour conferred upon him and stated that he would try to follow in the footsteps of his illustrious predecessors.

Nomination of Members.

The following were nominated for membership:

DR. A. E. COLVIN, Orange.
DR. R. B. NORTH, "Beanbah," Macquarie Street, Sydney.
DR. COLIN C. ROSS, "The Albany," Macquarie Street, Sydney.

Melanotic Growth.

DR. F. J. BLAXLAND presented a patient, a young woman, who had been hit with the heel of a shoe on her left cheek five years previously. Her cheek had bled a little; a black eye had followed. Soon afterwards she had noticed a small, pigmented patch between the limbus and the inner canthus of the left eye. In June, 1922, she had noticed that one part of the melanotic patch had become thicker and more marked than formerly. A curved, branching melanotic area was seen extending slightly into the cornea above and a thickened area in the upper part of the curve.

The case was discussed at some length by the members present. Various suggestions were put forward. DR. NORTH and DR. DUNLOP suggested the excision of a piece for pathological examination. DR. JONES suggested watching the mass closely for several months.

In reply to Dr. North, DR. BLAXLAND said he had thought of excision for pathological purposes, but had decided against it, as he wished to remove the growth and to save all the conjunctiva possible.

Hyaloid Degeneration of Blood Clot in the Vitreous.

DR. E. TEMPLE SMITH showed a patient whose case had been described in *The British Journal of Ophthalmology* of August, 1918, by Captain Viner. It was really a case of hyaloid degeneration of blood clot in the vitreous.

This case was discussed at some length. DR. JONES suggested that as the mass had been found a week after the blood clot, it was highly improbable that the change would have occurred in such a short time. He suggested that the hyaloid mass was congenital in origin.

Correspondence.

THE CONTROL OF VENEREAL DISEASE.

SIR: I notice in the current number of THE MEDICAL JOURNAL OF AUSTRALIA you have a leader on "The Control of Venereal Disease." Figures are quoted from the States of Victoria and of Western Australia. These figures appear to me to be grotesque.

Unless venereal diseases are treated by the medical profession alone and unless all cases are treated, figures are of little value.

I doubt if anything like 50% of the venereal population of either of those States are being treated by legally qualified medical practitioners.

Judging by this State (New South Wales), my concession would be high.

In New South Wales there is a steadily increasing percentage of venereal patients who are being treated by unqualified men or chemists. This I know by questioning patients, both in my private and in my hospital clinic.

One patient at the hospital clinic pulled a bottle containing a well-known oil used in the treatment of gonorrhœa out of his pocket and admitted having obtained it from a chemist without a prescription. He said he thought everything was all right as the chemist was a J.P.!

It would be advisable to strictly enforce the *Venereal Diseases Act* before we attempt to give any figures for this State. The figures might have some value then.

Yours, etc.,

J. COOPER BOOTH.

171, Macquarie Street, Sydney,
November 3, 1922.

DIATHERMY AND RADIO-THERAPY IN THE TREATMENT OF MALIGNANT GROWTHS.

SIR: Dr. W. Kent Hughes writes: "A rodent ulcer that has attacked periosteum, bone or cartilage, has only two methods of cure—surgery or diathermy."

Nevertheless, I have treated a patient who has lost practically the whole of his projecting nose on account of rodent ulcer with extensions into both eyes and of twenty years' duration by means of intensive X-ray therapy, using a voltage of 200,000. The first dose was administered on July 20 and the only other on September 8 of this year. Nevertheless, the whole lesion has practically healed, the eroded cartilage healed at once, being very soon covered with mucous membrane.

Dr. Hughes has seen this patient since treatment was started. Both surgery and diathermy have been out of the question, although the former will be of great value in fashioning a new nose.

Yours, etc.,

H. FLECKER.

4, Collins Street, Melbourne,
November 4, 1922.

SIR: In reply to Dr. Hughes's letter of October 23 I have to protest against his *tu quoque* statement with regard to prejudice on my part against diathermy in response to my reference of his obvious and well-known (I might almost say notorious) prejudice against radiation treatment. Those who knew me, will understand that this accusation is absurd, realizing that I am willing to discard any method of treatment for a better. Fortunately, my sceptical mind insists upon a critical examination of the *pros* and *cons* such as I have endeavoured to place before the readers of this correspondence in my previous letters. These comparisons are not attacked by Dr. Hughes, who simply draws a red herring across the scent by accusing me in turn of prejudice and repeating statements which I have shown to be ill-founded, *e.g.*, that one method (radiation) takes a long time, followed by a statement a little later "that, if limited to six months, little harm would be done beyond a waste of time and money." The

obvious implication involved is that neither is much good and Dr. Hughes knows that is not so.

The mutilation argument which I used, has no reference to the appearing of resulting scar, as Dr. Hughes well realizes, unless he failed to read the editorial in which this word was used. It applies, as I explained, to the destruction of healthy along with diseased cells by diathermy and surgical methods.

The usefulness of diathermy is very real one and I have had it done repeatedly for my patients, but my contention is that the statement that diathermy is preferable to radiation treatment in any but selected cases is incorrect.

Dr. Hughes persists in making bald but vague statements, *e.g.*, "that in his experience many intermediate cases are not cured." but that "many" may well be only a small proportion of the total cases so treated. I have shown that 90% of all cases—early, intermediate and late—can be cured. Then he says that once rodent has attacked periosteum, bone or cartilage there are only two methods of treatment—surgery and diathermy.

This used to be true, but is so no longer, the high voltage tube and short wave length ray being responsible for the change. Cartilage and periosteum have few terrors now and bone to the 200 kilo-volt tube is hardly less permeable than ordinary tissue. It may surprise Dr. Hughes that of the seventy-eight patients mentioned in my paper (THE MEDICAL JOURNAL OF AUSTRALIA, July 1, 1922) as cured, one had involvement of bone and at least seven involvement of cartilage.

As to the last paragraph, referring to frequence of awful cases of rodent, I can only say that Dr. Hughes's Melbourne experience is different to my Sydney experience and that the corroboration of his statement is more than counterbalanced by the corroboration of mine.

I have inquired of Drs. Godsall and Mansfield (Ear, Nose and Throat Department, Royal Prince Alfred Hospital), who are emphatic in their statement that these extensive cases are much rarer than in their student and resident days; Dr. Marsh (Ear, Nose and Throat Department, Sydney Hospital) expresses himself in similar terms; Dr. Warren (Sydney and South Sydney Ear, Nose and Throat Departments) also supports me; Sir Alexander MacCormick says that his impression is that such cases are noticeably less common than they used to be, owing to earlier treatment being instituted. This evidence could be multiplied indefinitely, if Dr. Hughes so desires, but space forbids.

Everyone that I have asked (not "more than one," as Dr. Hughes vaguely states) has supported my contention. Therefore, whatever Dr. Hughes himself believes, it is necessary for him to produce more evidence than he has hitherto thought necessary to support his contention and to persuade his readers that the latter is correct.

Possibly conditions in Melbourne may be different, though it is hard to realize, but certainly in the Royal Prince Alfred Hospital extensive use of radiation methods for the last fifteen years has produced a smaller and not a greater number of devastating rodents than before the institution of such methods.

Yours, etc.,

E. H. MOLESWORTH.

"Beanbah," 235, Macquarie Street, Sydney,
November 6, 1922.

THE ARSENO-BENZOL DRUGS.

SIR: In your journal of 21st *ultimo* Dr. H. Rischbieth refers in his article on "The Therapeutic Value of the Arseno-Benzol Drugs" to a gradual diminution in therapeutic efficiency during the past few years.

Similar observations have been made by many others, whose experience qualified them to express an opinion. The question is of much importance. Dr. Rischbieth ascribes the alteration to an effort on the part of commercial firms to cheapen the method of production. I think this does not quite represent the position.

Latterly, following the introduction of more soluble compounds, the profession has come to appreciate the additional convenience afforded and the freedom from toxic reactions, even when injection is made in highly concentrated solution. Thus the demand grew and to meet this the chief effort of manufacturers has been to supply preparations of ready solubility and of minimum toxicity.

It appears that arseno-benzol, as commercially prepared, by reducing nitro-hydroxy-arseno-benzine with hyposulphite, incorporates varying quantities of sulphur-containing compounds, as well as sulphur in the adsorbed state. Sulphur apparently diminishes the toxicity, since chemically pure arseno-benzol is both less soluble and more toxic than the commercial product.

These circumstances explain why variations of the manufacturing process have altered the character of the salvarsan compounds. Therapeutic efficiency can most conveniently be estimated by observation of the effect of the drug on trypanosomes in infected mice. In addition to the toxicity test at present carried out, a therapeutic test on the above lines is now being instituted by the Board of Trade in England and should result in inducing manufacturers to endeavour to approach the old standard of efficiency displayed by the original German preparation.

Yours, etc.,

C. J. WILEY.

203, Macquarie Street, Sydney,
November 6, 1922.

PUERPERAL INFECTION.

SIR: Dr. Chenhall states that his remarks applied to obstetrical operations, but, if his figures be correct, it is difficult to mention any more fatal operation than manual removal of the placenta, obstetrical or surgical.

He quotes statistics to prove a mortality of 13% in one series of cases and 20.8% in another. To my mind this is ridiculous and shows how figures may be used to prove anything. We only want to get at the truth and, if there be an increased mortality of 0.001% owing to manual removal, such interference is inadmissible. I feel satisfied, however, that the truth does not dwell in the figures given, wherever they come from.

In the face of such an appalling mortality, any intrauterine manipulation after parturition would be unjustifiable without regard to "personal skill and good fortune."

The point which I was trying to establish, was that there is a time in puerperal infection when it is possible to control it. As with a bush-fire, a bucket of water used early enough would suffice to stop it. So I believe there is a time when the gauze and iodine application previously referred to will extinguish uterine infection, if we can only avail ourselves of that time. The problem is simplified to the extent that we know the place of greatest danger.

It is usually stated that antiseptics have disappointed us in the treatment of puerperal sepsis, but I content that they have not been given a fair chance in that they are constantly used too late.

It is useless applying antiseptics to the uterine cavity when the infection has passed into the sinuses or beyond. If we acted on the very first indication of sepsis, there would be a good chance of overtaking the enemy.

With regard to asepsis, Dr. Chenhall has quite failed to grasp my meaning. I repeat that, while asepsis is all-important in preventing heterogenous infection, such as the carriage of infection from one case to another by the hands or instruments, it is practically impossible to insure asepsis of the patient's skin surfaces—vulva, vagina, urethra, anus and perineum. Moreover, the secretions from these parts will cause re-contamination during the progress of delivery.

Autogenous infection, therefore, is always possible, even wtih the greatest care provided; I would add, a culture medium is present.

Dr. Chenhall thinks that bacteria, if carried in, can attack an empty uterus equally with one containing retention products. On this point we are not in agreement. I believe that lochial stasis is very often present when the discharge is apparently free and this pabulum for germ growth is easily overlooked.

In the Isolation Block of the Women's Hospital, Crown Street, I have over and over again demonstrated to students

the presence of retained lochia in cases of sepsis where the uterus was supposed to be empty.

Granted the evil effects of impatient and meddlesome midwifery. Cases of puerperal infection are common and often it arises in cases where there has been no interference whatver. The question is: "Can we do anything better in the way of treatment when it has occurred?" I think we can, if we became more keenly alive to the importance of the time factor and less fearful of investigating the uterine cavity.

Yours, etc.,

J. MORTON.

Macquarie Street, Sydney,
November 7, 1922.

Proceedings of the Australian Medical Boards.

NEW SOUTH WALES.

THE resignation of DR. H. J. W. BRENNAND of his position as Member of the New South Wales Medical Board has been announced in the *Government Gazette of the State of New South Wales*, No. 155, of November 3, 1922.

Books Received.

A MANUAL OF PRACTICAL ANATOMY, by Thomas Walmsley; Part III., The Head and Neck; 1922. London: Longmans, Green & Company; Demy 8vo., pp. 272, with 96 figures. Price: 10s. 6d. net.

AN OUTLINE OF THE MEDICAL SERVICE OF THE THEATRE OF OPERATIONS, by M. A. W. Shockley, Lieutenant-Colonel, Medical Corps, United States Army; 1922. Philadelphia: P. Blakiston's Son & Company; Post 8vo., pp. 230, with eight detached diagrams of field operations. Price: $2.50 net.

MODERN METHODS IN THE DIAGNOSIS AND TREATMENT OF GLYCOSURIA AND DIABETES, by Hugh Maclean, M.D., D.Sc., M.R.C.P.; 1922. London: Constable & Company, Limited; Sydney: Angus & Robertson, Limited; Demy 8vo., pp. xi. + 159, with thirteen charts and nine figures. Price: 12s. 6d. net.

PHYSICAL DIAGNOSIS, by W. D. Rose, M.D.; Third Edition; 1922. St. Louis: C. V. Mosby Company; Royal 8vo., pp. 755, with 319 illustrations. Price: $8.50 net.

THE ELEMENTS OF SCIENTIFIC PSYCHOLOGY, by Knight Dunlap, Professor of Experimental Psychology in The Johns Hopkins University; 1922. St. Louis: C. V. Mosby Company; Crown 4to., pp. 368, with 31 illustrations. Price: $.350 net.

NOTE.—Our attention has been directed to the fact that the English edition of the book entitled "Practical Bacteriology, Blood Work and Animal Parasitology," by Dr. Stitt, of which a review was published on July 29, 1922, is issued by H. K. Lewis & Company, Limited, and that the English price is 20s. net.

Medical Appointments.

DR. A. E. ROWDEN WHITE (B.M.A.) has been appointed to the position of Honorary Physician to In-Patients at Saint Vincent's Hospital, Melbourne.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii..

ADELAIDE CHILDREN'S HOSPITAL (INCORPORATED): Resident Medical Officer.

MELBOURNE HOSPITAL: Vacancies on the Honorary Staff.

ROYAL HOSPITAL FOR WOMEN, PADDINGTON: Honorary Assistant Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney	Australian Natives' Association Ashfield and District Friendly Societies' Dispensary Balmain United Friendly Societies' Dispensary Friendly Societies Lodges at Casino Leichhardt and Petersham Dispensary Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney Marrickville United Friendly Societies' Dispensary North Sydney United Friendly Societies People's Prudential Benefit Society Phœnix Mutual Provident Society
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne	All Institutes or Medical Dispensaries Australian Prudential Association Proprietary, Limited Manchester Unity Independent Order of Oddfellows Mutual National Provident Club National Provident Association
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane	Brisbane United Friendly Society Institute Stannary Hills Hospital
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide	Contract Practice Appointments at Renmark Contract Practice Appointments in South Australia
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth	All Contract Practice Appointments in Western Australia
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington	Friendly Society Lodges, Wellington, New Zealand

Diary for the Month.

Nov. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 21.—Illawarra Suburbs Medical Association, New South Wales: Annual Meeting.
Nov. 23.—Brisbane Hospital for Sick Children: Clinical Meeting.
Nov. 24.—New South Wales Branch, B.M.A.: Branch.
Nov. 24.—Queensland Branch, B.M.A.: Council.
Nov. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee; Organization and Science Committee.
Nov. 28.—Victorian Branch, B.M.A.: Ballot Paper Issued.
Nov. 29.—Victorian Branch, B.M.A.: Council.
Nov. 30.—South Australian Branch, B.M.A.: Branch.
Dec. 1.—Queensland Branch, B.M.A.: Branch.
Dec. 5.—New South Wales Branch, B.M.A.: Ethics Committee.
Dec. 5.—Victorian Branch, B.M.A.: Ballot Papers Returned.
Dec. 6.—Victorian Branch, B.M.A.: Annual General Meeting.
Dec. 8.—New South Wales Branch, B.M.A.: Branch.
Dec. 8.—Queensland Branch, B.M.A.: Council.
Dec. 8.—South Australian Branch, B.M.A.: Council.
Dec. 9.—Eastern District Medical Association, New South Wales.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

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